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HOSPITAL

OF

THE PROTESTANT EPISCOPAL CHURCH
IN PHILADELPHIA

MEDICAL AND SURGICAL
REPORTS

OF THE

EPISCOPAL HOSPITAL

VOLUME VI

Commemorating the Seventy-fifth Year of the Hospital

PHILADELPHIA

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1930

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1852-1927

This volume commemorates the seventy-fifth year of the hospital's existence. For this reason special articles reminiscent of the early days of the hospital have been included. Of the remaining articles some have previously appeared in various medical journals, being here reprinted through the courtesy of the Editors of those journals, while others have been prepared especially for Volume VI.

The Medical Staff desire to express their high appreciation of the liberality of the Board of Managers of the Hospital in making provision for the continuance of the publication of this series of volumes.

The Committee of Publication think it proper to say that the Hospital holds itself in no way responsible for the statements, reasonings or opinions set forth in the various papers published in this volume.

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RALPH S. BROMER, M.D.,
ASTLEY P. C. ASHHURST, M.D., *Chairman.*

This volume has been edited by

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Bulletin de l'Académie Royal de Médecine de Belgique. Bruxelles, Belgium.
John Crerar Library Publications. Chicago, Illinois.
Crouse-Irving Hospital Bulletin. Syracuse, New York.
Johns Hopkins Hospital Bulletin. Baltimore, Md.
Massachusetts General Hospital, Collected Papers. Boston, Mass.
Münchener medicinische Wochenschrift. Munich, Germany.
Philippine Journal of Science, Medical Section. Manila, P. I.
Pilcher Hospital, Bulletin of the. Brooklyn, N. Y.
Presbyterian Hospital Reports. New York, N. Y.
Roosevelt Hospital, Medical and Surgical Reports. New York, N. Y.
Royal College of Surgeons of England, Calendar. London, England.
University of Missouri Bulletin, Medical Series. Columbia, Mo.
Westminster Hospital Reports. London, England.
Wiener klinische Wochenschrift. Vienna, Austria.

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FROM THE SUPERINTENDENT'S REPORTS OF 1853 AND 1928



	Number of Patients	
	1853	1928
Remaining in hospital January 1st	212
Remaining in Harrison Home January 1st	78
Admitted during the year	180	6,402
Admitted to the Harrison Home	13
Total number treated during the year	180	6,705
Of these there were:		
Discharged, recovered	103	3,534
Discharged, improved	27	2,241
Discharged, unimproved	10	76
Died	22	529
Remaining in hospital December 31st.	18	325
Total	180	6,705
Average: days in hospital not including Harrison		
Home	31.7	13.3
Average daily number of patients in hospital	318
The largest number treated on any one day	369
The smallest number treated any one day	260
Percentage ward patients	87
Percentage private patients	13
Cost per day for ward patients	\$3.82
Total number of ambulance calls	6,380
Total number of patients treated in Dispensary and Emergency Department	31,901
Total number of visits of patients to the Dispensary and Receiving Ward	98,166
Average cost per out-patient visit	\$0.66
Average daily number of patients treated in all depart- ments	588
Average daily number of persons supported	703

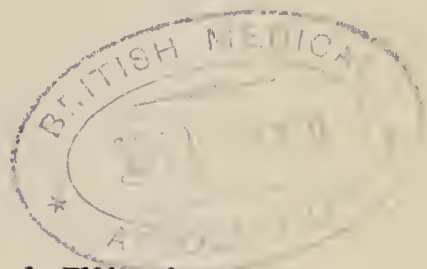




RICHARD H. HARTE, M.D.

RICHARD HICKMAN HARTE*

BY ASTLEY P. C. ASHHURST, M.D.,
SURGEON TO THE HOSPITAL



RICHARD HICKMAN HARTE was born in Rock Island, Illinois, October 23, 1855. He passed his entire professional life in Philadelphia and died, November 14, 1925, at Vicksburg, Mississippi.

Dr. Harte was graduated from the Medical Department of the University of Pennsylvania in 1878, and received his early training in surgery in the University Hospital as assistant to Agnew and to Ashhurst; and later in the Pennsylvania Hospital, where he became a surgical chief in 1893. He also served as surgeon to the Episcopal Hospital (1889-1904), to St. Mary's Hospital (1893-1899), and to the Orthopædic Hospital (1904-1914).

Possessed of an ample fortune, Dr. Harte may be said to have practised his profession as Johann Sebastian Bach wrote music, "for the glory of God and for a pleasant occupation." He never had a very large private practice, but delighted in his work in the hospital wards, paying particular attention to the old, the helpless and the miserable, especially to those unfortunates whose sojourn is long in the dreary dwellings which border on the shades of death. Though never of very robust physique himself, he radiated an atmosphere of cheerfulness and hope among his patients and they cherished his visits and appreciated his neatness and gentleness in dressing their wounds more than his operative skill, of which they knew nothing.

Between the ages of fifty and sixty years, Dr. Harte gradually withdrew from practice and resigned, one after another,

* Taken from the article entitled, "In Memoriam, Richard H. Harte, M.D., C.M.G., Hon. F.R.C.S. (Ire.), 1855-1925." By Astley P. C. Ashhurst, M.D. Transactions of the American Surgical Association, 1926. Reprinted from Surgery, Gynecology and Obstetrics, 1929, xlviii, 135.

all of his hospital appointments except that of surgeon to the Pennsylvania Hospital.

Elected a Fellow of the American Surgical Association in 1895, he soon became an active and interested member, rarely missing the annual meetings and contributing a number of valuable papers to its *Transactions*. From 1900 to 1909, he served as recorder, until his election as president of the association in 1910.

Of Dr. Harte's war service, it is impossible to speak adequately. Feeling very strongly the call of duty to assist the Allies, he left his home and his many engagements in this country early in 1916 and served for many months in the American Hospital at Neuilly-sur-Seine (Paris). Returning to Philadelphia in the autumn of 1916, and foreseeing the entrance of the United States into the conflict, he set about organizing two base hospital units, one in connection with the Pennsylvania Hospital, which became Base Hospital No. 10, A.E.F.; the other in connection with the Episcopal Hospital, which became Base Hospital No. 34, A.E.F. As director of Base Hospital No. 10, he left for France in the spring of 1917, and remained on active duty in France until after the armistice. During his absence he had the greatest sorrow of his life, the death of his wife, but he sought to forget his grief in constant activity, saying that he knew it would be her wish for him to complete the task he had undertaken, and he never faltered. He was rapidly promoted to the rank of colonel, and illness alone prevented him on his return to this country in the winter of 1918 from serving as chief surgeon of the Walter Reed General Hospital, Washington, D. C.

He received from General Pershing a citation for "exceptionally meritorious and conspicuous service." His work with the British Army was of such importance that it was mentioned in dispatches by General Haig, and later Dr. Harte was made a companion in the British Order of St. Michael and St. George. The King of the Belgians decorated him as companion in the Order of Leopold, and he was made honorary fellow of the Royal College of Surgeons of Ireland for "conspicuous service rendered to the British Expeditionary Forces." From our own country, he received the Distinguished Service Medal.

Dr. Harte wrote very little. He took special interest in

the surgical complications of typhoid fever, and published a number of important papers on perforation of, and hemorrhage from, the intestines during that disease. He had probably the largest personal experience of such complications of any surgeon in the world.

Dr. Harte had a rare intuition of diagnosis and prognosis, a surgical judgment which was almost infallible, and operated with an ease and deftness which I have never seen equaled either in this country or abroad. Every scalpel that he used seemed sharp, tissues fell asunder as if by magic and with nearly complete absence of bleeding, ligatures dropped from his fingers as if already tied, and wounds healed with the most surprising rapidity and with the minimum of scarring. It was with the deepest regret that his assistants saw him abandon his career as operating surgeon so comparatively early in life. He resumed it only for a short time in France during the World War, and was delighted that the first patient on whom he operated at the front, a soldier with multiple gunshot perforations of the bowel, made an excellent recovery.

He was, as I have said, never robust. Subject to bronchial inflammations, he rarely passed a winter without being in bed for a few days on one or more occasions. He also suffered a good deal from a stiff and painful shoulder, due to what he called "neuritis," which made him miserable sometimes for weeks at a time. When he was run down, nothing would restore his health so soon as a river trip or coasting expedition, either in his own yacht or in that of his bosom friend, Dr. William J. Mayo.

Leaving Philadelphia with a bad cold and with his arm in a sling, on November 2, 1925, he joined Dr. Mayo on his boat on the Mississippi River two days later at Memphis, had a severe chill, and took to his bed at once. As soon as the presence of pneumonia was suspected, he was transferred to the hospital at Vicksburg, and there, on the shores of his favorite river, after a brave fight against the disease for more than a week, attended by Dr. Mayo and other friends, and with his children beside him, his spirit passed on to the other shore of the river of death. He had lived his life with a conscience void of offense toward God and toward man.

A SKETCH OF EARLY MEDICINE AT THE EPISCOPAL HOSPITAL

By ELLISTON J. MORRIS, M.D.,
CONSULTING PHYSICIAN TO THE HOSPITAL

"THE Hospital of the Protestant Episcopal Church in Philadelphia" owes its origin to Bishop Alonzo Potter, who saw the urgent need of more accommodation for the sick and injured than was then available, and on March 14, 1851, called a meeting of the influential men of the Diocese for the purpose of founding such an institution. At that time the population of Philadelphia is estimated to have been approximately 400,000 and there were but four hospitals—the Pennsylvania, founded about a century previously, the City Hospital at Blockley; the Wills Eye Hospital, and St. Joseph's Hospital, then lately begun at "Green Hill," now Sixteenth and Girard Avenue. Due to the efforts of Bishop Potter's committee, the daughters of Mr. John Leamy presented a part of their estate of "Tusculum" as a site for the Hospital, and this gift of about six acres was added to by the purchase from the Leamy Estate of a sufficient amount to complete the present holding. The deed of gift is dated the ninth day of July, One Thousand Eight Hundred and Fifty-two, and the work of alteration of the Leamy Mansion for the reception of patients was immediately begun and the Hospital was formally opened on December 11, 1852. The first patients were not received, however, until December 24, 1852, at which time four members of the same family were admitted, "suffering from a fever." Accounts vary as to whether this fever was typhus fever or typhoid fever, and as the distinction was not sharply drawn between the two diseases at that time, it is probable that the patients suffered from typhoid.

It was only a very short time before the accommodations of the Leamy Mansion proved too scanty for the work of the

Hospital and the necessary funds were raised for the beginning of the present group of buildings. The oncoming of the Civil War delayed their completion, but the demand of the Government for hospital accommodation for sick and wounded soldiers finally hastened the work, and in July, 1862, the buildings then completed (which consisted of the present men's wards and the chapel) were temporarily turned over to the Government and were used as a military hospital until March, 1863, when other hospitals were erected by the Government and the Episcopal Hospital was returned to the uses for which it had originally been intended. From time to time other wards and other buildings have been added, until the institution stands as it does today.

It has added greatly to the difficulties of preparing this sketch that the early records of the Hospital would seem to be missing to a large extent and the available early records would seem to consist mostly of a list of admissions and discharges, with only the diagnoses given, and little or nothing said as to treatment. The scarcity of early records is probably due to two causes: some of the records were unfortunately burned in a fire in a printing house to which they had been sent for binding; probably the case histories were, in most instances, recorded by the visiting chiefs and were retained by them as their own personal property for use in preparing medical papers and in this way have been lost to the Hospital. This last is mere theory, but the fact remains that the earliest records which the writer has been able to obtain are those made in 1879, during the service of the late Dr. Frederick P. Henry. No doubt there were earlier records, but they would seem to have perished in the fire in the binder's shop or to lie hidden, waiting for some more fortunate lover of the past of the Hospital to discover them.

In the absence of definite records one can only try to build the structure from the fragments which we possess and from the works on the practice of medicine of the period of 1858 and 1860; but the writer feels that the result will be fairly accurate, since the staff of the Hospital has been always composed of men who were eager to give the Hospital the best medical practice of their day.

Bloodletting, tartar emetic, opium, calomel or blue mass would seem to have been the sheet anchors of the practitioner of that day. It is advised that in the early stages of pneumonia the patient be bled to the extent of one or two pints, if he is strong enough in the opinion of his attending physician; tartar emetic follows the bleeding, and later in the course of the disease "digitalis may be necessary to support the heart." One shudders at the nauseous draughts administered. Decoctions, infusions and tinctures were the order of the day. Pills were bulky and hard to swallow. Capsules, compressed tablets and the small modern pill had not yet been devised. Hypodermic medication was unknown and the writer was astonished to hear Dr. W. W. Keen say that he doubted if there were more than three or four hypodermic syringes in the entire Northern army during the war from 1861 to 1865. The writer has in his possession one of the earlier forms of hypodermic syringe, in which the piston-rod is threaded like a screw, so that by screwing down the piston the liquid can only be slowly forced under the skin; this construction being apparently caused by the fear of suddenly injecting the contents of the syringe barrel. One really admires those old practitioners; they seem to have done so much with so little to work with. The clinical thermometer did not come into use until a later period, and the physician judged of the patient's temperature (degree of fever) by the state of the pulse and the doctor's sense of touch. A recent pamphlet on the clinical thermometer states that the "first record we have of the use of the thermometer in America in cases of fever was that of Dr. Billings, who had charge of the wounded in the seven days' siege before Richmond in 1862. Prior to 1864 the thermometer was entirely unsuited to the taking of human temperature, being about a foot long, took several minutes to register and had to be carried in a holster." The stethoscope was a straight tube of wood or metal, with a large bell at one end to apply to the doctor's ear and a smaller bell at the other end for application to the chest of the patient. The binaural stethoscope did not come into general use until about 1883 or 1884.

Laboratory work was chiefly confined to the examination of

urine, but there were no centrifuges and the sediment was allowed to collect in the bottom of a conical glass and examined after a lapse of twenty-four hours, by which time the examination was made unpleasant by the odor and many casts destroyed by the putrefaction which had set in. Blood examinations, as at present understood, were totally unknown. There were two blood corpuscles, *the* red and *the* white; the important rôle of the leukocyte was unrecognized. All laboratory work was done by the resident physician in charge of the ward in which the patient lay and there was no clinical laboratory such as we know today. In the report of the Board of Managers for the year 1898 (issued January, 1899) appears this note:

“CLINICAL LABORATORY. The Board recognizes that it is important for an Institution of the magnitude of this Hospital to have a well-appointed clinical laboratory, and it stands ready to establish such a laboratory whenever the necessary funds shall be provided for the purpose.”

Just when the Laboratory was established the writer is not able to definitely state, and the question is perhaps beyond the scope of the present sketch; but apparently it was started shortly after 1899, under the care of Dr. William Egbert Robertson (for long a valued member of the medical staff).

While the above remarks in regard to the clinical investigation as a specialized department are true, it must not be thought that the profession was lethargic, nor that our institution was not alive to its duties to its guests. Dr. Frederick P. Henry, physician to the Hospital from 1874 to 1888, was, for example, contributing important papers to medical societies, and his work on the *filaria sanguinis hominis* is of especial importance. Everywhere there was an immense amount of research work quietly going on, to culminate in Koch's announcement of the bacillus tuberculosis as the cause of phthisis (1882); his discovery of the cholera bacillus (1884), and the growing knowledge of the rôle of microorganisms as causative factors of disease. Modern medicine was in the making during all these years of patient, plodding toil.

This revolution in pathological thinking was not brought about without bitter controversy. The writer well remembers the sharp debate which took place between Dr. Henry F. Formad, of the University of Pennsylvania, and Koch, of Berlin. Formad claimed the tubercle bacillus to be the result of tuberculosis and Koch stood his ground that the germ was the cause. Formad claimed that he could produce tuberculosis in white mice (a tuberculous animal anyway, said Formad) by putting ground-glass from his laboratory into their abdominal cavities. Undoubtedly he did produce tuberculosis in this way, but the sterilization of the glass was crude, or none at all, and the bottles of the laboratory from which the ground-glass was made were reeking with tubercle bacilli and all the other forms of bacterial life.

Gradually the new pathology gained over the old and medical thought turned to remedies which would kill the microorganisms, only to be disappointed by finding that any germicide strong enough to be of service was as harmful to the body cell as to the germ cell. Remedies which now seem strange had their day of fashion. Tincture of iodine was used for a time in the treatment of typhoid fever, with the idea of a germicide in mind. Here is a transcript from the treatment sheet of a case of typhoid fever in 1887: "Acid carbolic gtt. i, Tinct. Iodi gtt. i every three hours," and this treatment was continued for nine days. The patient apparently recovered. In another case of typhoid the carbolic acid was given in three-drop doses every three hours for seventeen days; in this case also the patient apparently recovered. Enemata of sulphuretted hydrogen were given in tuberculosis, since sulphur was long thought to be of benefit in phthisis. The patients expressed themselves as being benefited by these enemata, but the utter folly of the treatment soon made itself manifest.

Perhaps we have gone ahead of the story. Let us go back to those old records by Dr. Henry in 1879; they are interesting. Milk-punch or some form of alcoholic stimulation seems to have been given to every patient, as far as one can tell. In the hospital report for 1884 the bill for brandy and spirits was \$832.94; wine cost \$129.20; porter and ale were not so popular, apparently, and only cost \$67.20 for the 1727 patients cared

for in the wards during that year. In 1923 (the last year in which the writer can find the cost of wine listed in the reports) the wine bill was \$288.60 for the 5847 patients cared for; brandy and spirits have ceased to be separated from the bill for alcohol in general.

Peritonitis was treated by giving $\frac{1}{4}$ to $\frac{1}{2}$ grain of morphia every hour until the patient was thoroughly narcotized; turpentine stupes were applied to the abdomen. The surgeon feared to invade the abdomen. Peritonitis was a disease, not a symptom.

Here is a case diagnosed as "typhlitis" (inflammation of the colon). A sharp diagnostic difference was drawn between typhlitis (inflammation of the colon) and perityphlitis (inflammation around the head of the colon); but I strongly suspect that many a case of appendicitis masqueraded under this diagnosis. Appendicitis is not named in the index to Pepper's *System of Medicine* (1885), nor in Agnew's *Surgery* (1883). The treatment given was a pill of 1 grain of powdered opium and $\frac{1}{2}$ grain of blue mass three times a day, and enemata of warm water. Three days later the treatment was changed to a pill of 1 grain of compound extract of colocynth and $\frac{1}{2}$ grain of extract of belladonna three times a day, with two enemata of soap and warm water per day. Apparently the patient recovered, since there is no note to the contrary.

In 1888, as resident on the Men's Surgical Ward, it was the writer's good fortune to have under his charge one of the earliest operation cases of appendicitis. Dr. Charles B. Nancrede, who was the surgeon on duty at the time, claimed that it was the first case in Philadelphia where the diagnosis of appendicitis was made before the abdomen was opened. A few cases had been operated on at the Pennsylvania Hospital and appendicitis found at the operation, but Dr. Nancrede always insisted that this was the first operation with the deliberate purpose of removing a diseased appendix. The patient was an utterly worthless Kensington tough and was admitted under the diagnosis of typhlitis. After two days' stay in the medical ward he was seen by Dr. Nancrede in consultation with the medical chief, transferred to the surgical ward and operated on. In spite of the delay in operation, and

also in spite of the fact that he wore a glass drainage-tube for about three weeks, he finally recovered. It was the writer's duty to pump out that drainage-tube every two hours (later, every three hours), day and night, during the time in which it was in place, because there were then no nurses to whom the duty could be intrusted; and this brings us to the subject of the nursing staff of the Hospital—a very important part of the care of the sick, but perhaps a little aside from the main purport of this sketch.

In its original charter the Hospital had declared that one of its duties should be the training of suitable persons for the care of the sick; but no training school for nurses, as we now understand such, was started until the beginning of the year 1888. Previous to that time the nursing had been done by women in the female wards and men in the male wards, and it was a question as to whether or not it was right to ask a woman to care for a male patient. Under the régime in vogue prior to 1888, these nurses remained in the hospital for an indefinite period—some of them spent practically their entire lives in the wards until they grew so old that they were no longer useful and were pensioned off by the Hospital. Others gathered some knowledge of nursing from their work at the Hospital and went away to do such private nursing as they could obtain. There was no course of training—there were no classes—there was no graduation. One stayed as long as he wished, gathered as much knowledge as he could and then went out as a nurse and said he had been a nurse at the Episcopal Hospital and that was all that there was about it. But there is another side to the picture. Those old nurses gave their lives up to the care of the sick and injured and did it with often a high altruistic motive and with little thought as to the commercial side. They lived for years in the hospital; gradually they acquired a keen insight into the probable course of the disease and their advice was often useful and instructive to us as newly graduated resident physicians. The contemporaries of the writer will remember old Abram, the night-nurse on the Men's Medical Ward; Hopkins on the Men's Surgical Ward. There were others, but their names have slipped into oblivion though their kindly advice and council

to the young resident still comes to his mind though the years have gone by since then and many nurses passed through the training-school.

Times change and we change with them, said the old philosopher. We smile or frown, as the humor takes us, over the tartar emetic for pneumonia or the sulphuretted hydrogen for tuberculosis. Our children will probably smile or frown, as the humor takes them, over what seemed the truth to us. But, with the acquisition of modern diagnostic aids—the x-ray, the electrocardiograph, the laboratory—have we not lost some of that keen observation and almost uncanny diagnostic insight of our forebears?

I have said nothing as to the personnel of the staff of the Hospital during these early years, for the reason that it seemed that this should be the subject for a separate memoir by some one better qualified than the writer. Always they were men of high standing. Always they worked as if they had in mind the words of one of them, uttered at the turning of the first sod for the larger buildings in 1860: "*In nomine Patris, et Filii, et Spiritus Sancti, hospitium instituo, ad majoram dei gloriam salutemque generis humani.*" (In the name of the Father, and of the Son, and of the Holy Ghost, I begin this hospital, to the greater glory of God and the welfare of mankind.)

INCREASE IN COST OF HOSPITAL ADMINISTRATION DURING THE LAST SEVENTY-FIVE YEARS

By CHARLES A. GILL, PH.G.,
SUPERINTENDENT OF THE HOSPITAL

It seems especially fitting that the question of cost should be considered in the publication of the Medical and Surgical Reports of the Hospital marking its Seventy-fifth Anniversary.

What is the difference between the total cost of maintenance in 1853 and that of 1928? Just a trifle over \$500,000, and the cost *per capita* increased during that period from 56 cents to \$3.82. This increase in cost has evidently been a matter of constant concern to our Managers, for as early as the fifth Annual Report (1857) is found a paragraph: "So greatly has the cost of all necessities of life advanced during the past and preceding year that it is absolutely impossible for us to avoid a large increase in expenditure."

The following table indicates the rapid increase in the cost of hospital care:

	Average number of patients under treatment.	Average daily per capita cost.	Average number of days under treatment.
1855 to 1865	33	0.56	30 minus
1865 to 1875	101	0.98	39 plus
1875 to 1885	125	0.99	38 plus
1885 to 1895	183	1.03	33 plus
1895 to 1905	258	1.15	26
1905 to 1915	315	1.46	20 plus
1915 to 1925	326	2.48	22 plus
1925 to 1928	319	3.28	17 plus

The cost per patient per day in certain different classes of hospitals cannot be compared; any comparative statement of

such *per capita* cost is of little value. A separation of departmental cost, for purposes of comparison, is of greater value. Until recent years there has been no cost-accounting system that made an attempt to separate the various department expenditures; it is merely a conjecture whether or not it is fair to assume that the cost of medical and nursing care has advanced in the same proportion as has the full cost of hospital care. In the year 1925, however, the Board of Managers installed a system of cost accounting that has given us an accurate account of the cost of various departments, thus enabling us to confine our consideration exclusively to the cost of medical and nursing care of the patient.

STATEMENT OF THE MEDICAL AND NURSING COST OF PATIENTS
DURING FOUR YEARS

	Cost of medical and nursing care of all patients.	Cost of medical and nursing care of in-patients.	Medical and nursing per capita cost	Cost of medical and nursing care of out-patients.	Cost per patient per visit.
1925	\$136,895.45	\$107,147.45	0.92 minus	\$29,748.00	0.31 minus
1926	145,758.40	111,757.43	0.96	34,000.97	0.37 plus
1927	150,823.48	111,814.62	0.95	39,008.86	0.40 plus
1928	163,113.58	121,753.01	1.04	42,360.57	0.43 plus

This brief statement indicates that the cost of medical and nursing care has increased from \$0.92 in 1925 to \$1.04 in 1928, or 13 per cent, and the cost for dispensary patients per visit shows an increase of \$0.31 in 1925 to \$0.43 in 1928, or 38 per cent.

Additional appropriations have recently been made from capital and depreciation funds amounting to more than \$19,000 for the purchase of new equipment, such as an electrocardiograph, a new basal-metabolism apparatus, x-ray and dental equipment, an electrosurgical unit, ether and oxygen apparatus, oxygen and carbon dioxide inhalators, physiotherapy and other equipment.

More liberal use is made of the pathological and x-ray laboratories, and of biological products and expensive drugs than in the past. Undoubtedly the age when patients were drenched

with drugs, when we could see only pills and potions, is fast passing; and no one appreciates this more than the writer, who has had personal experience in the old time apothecary shop, and spent days in rolling pills and hammering crude drugs into proper form to extract their principle by percolation. Although we must appreciate the higher standard of service attained in the practice of medicine, yet it is with feelings mingled with regret that we are forced to the realization of the apparent gradual passing of the United States Pharmacopœia and the increasing use of the formulary of various manufacturers of especially compounded and more costly pharmaceutical preparations. Then, too, the demand for costly medical aids and mechanical devices is constantly increasing in connection with the departments of radiotherapy, physiotherapy, dentistry, basal metabolism and electrocardiography.

While all this has added tremendously to the cost of operation, much has been accomplished in the reduction of patients' disability and their earlier return to society and work. As indicated in a study of the statistical reports of the hospital, the stay of patients has been reduced during the last decade from thirty days in 1919 to seventeen days in 1928, thus adding significantly to the productivity and earning power of the patient.

Modern preventive medicine has undoubtedly aided considerably in keeping down the cost of sickness through the aid of vaccination, toxin-antitoxin, purification of water, disposal of sewage and intensive campaigns against animal carriers. All this means a lower death-rate and reduced sickness. While the medical profession has made rapid progress in the practice of medicine, we are forced to ponder as to whether or not the administrative and managerial staffs of hospitals have taken full advantage of the many other forces known to assist in effecting cures by developing a more healthy hospital, its chief curative agents being rest, quiet surroundings, free ventilation, solariums, open air porches, ample air space and quiet rooms.

TYPHOID FEVER

By JOHN H. ARNETT, M.D.,
PHYSICIAN TO THE HOSPITAL

THE early years of the hospital were coincident with great advances in scientific knowledge relative to typhoid fever. Until 1837 it was not known, for example, that typhoid and typhus were separate entities: then, following the lead of Lombard,¹ W. W. Gerhard,^{2,3} of Philadelphia, showed from his observations upon patients in the Pennsylvania Hospital that these two infections could be both clinically and pathologically differentiated. In the following year Stillé, Gerhard's pupil, read before the Medical Society of Observation of Paris his "Table of Comparison between Typhus and Typhoid Fevers," so that Gerhard's work began to be known not only in this country but also in France. Next the mode of infection was brought to light by Budd in the *Lancet* in 1856-1860, and in 1880 Eberth discovered the organism causing typhoid.

The records of the Episcopal Hospital indicate that during the first twenty years of the hospital, that is from 1854 to 1873 inclusive, of 6001 medical cases admitted to the wards, 307 (or 5 per cent) were diagnosed typhoid fever. Of these, 54 (17.6 per cent) died. Bad as this mortality now seems to us, Gerhard wrote that of the patients under treatment for typhoid and typhus fevers in the famous clinic of Chomel in Paris, one out of every three died. Although the records of these early days are not sufficiently complete to enable us to say what treatment was administered, it seems probable that cold baths were not used in the majority of these cases, inasmuch as Dr. Brand, of Stettin, did not begin to popularize this treatment until 1861. The probabilities are that the treatment of typhoid in the Episcopal Hospital followed the same general lines as treatment elsewhere, including cupping, leeching,

bleeding and restriction of diet. Gerhard, for example, advised among other measures, "at the beginning, one or two moderate general bleedings; topical depletion by cups, or leeches to the anus, if the violence of the local symptoms and the strength of the patient indicate it."

The latest year for which bound histories of the Episcopal Hospital are available at the time of writing is 1923. During the eleven years from 1913 to 1923 inclusive, 413 cases diagnosed typhoid fever were admitted to the medical wards of the hospital. Of these, 45 died, making a mortality of 10.9 per cent. Of those who died, 7 (16 per cent) had symptoms of perforation, 15 (33 per cent) had intestinal hemorrhages, 16 (36 per cent) died of toxemia, cardiac failure or pneumonia, 6 (13 per cent) died of other conditions (decubitus, abscess, parotitis, cholecystitis, Ludwig's angina and influenza), and in 1 (2 per cent) the data are not sufficiently complete to indicate the cause of death. Thus, of the 413 typhoid fever cases studied 1.7 per cent died with symptoms of perforation and 3.6 per cent died with hemorrhages from the bowel. Various types of treatment were used (sponging, tubbing, vaccines and expectant), and various types of feeding were used (milk, reinforced milk, house diet). This mortality-rate, while a decided improvement over the 17.6 per cent of the early days, could, I believe, be still further lowered by the routine employment, during the more febrile stages of the disease, of tepid sponges and a diet of milk or modified milk. One institution in this city, where sponging and a milk or modified milk diet have been employed routinely for many years, had the remarkably low mortality of 5.6 per cent (178 cases with 10 deaths) during the years 1913 to 1923 inclusive. It must be recognized, of course, that there are other factors than the treatment which influence mortality; age, social status, habits of life and many other factors doubtless enter into the situation. No specific cure for the disease is known.

The greatest advance with respect to typhoid fever in the past seventy-five years has been not in its treatment, but in its prevention. Older physicians will vividly recall the wards and even the corridors of the hospital crowded with typhoid fever patients. Today we go through much of the year without a

case in our wards, so that our residents sometimes complain that they do not see enough typhoid to make them thoroughly familiar with the disease and its complications. This tremendous drop in the incidence of typhoid fever has been due almost entirely to the purer water supplied to our city. Lately, however, there have been intimations that the present sources of Philadelphia's water supply are more badly contaminated than is considered permissible according to modern standards. While the typhoid rate has thus far been maintained at a low level through the efficient operation of the municipal chlorinating and filtration plants, yet, if we do not wish to run the danger of another typhoid epidemic, it behooves us to heed the advice of sanitarians and find less contaminated sources of supply, so that we may no longer depend solely upon the efficiency of chlorinating and filtration plants to protect us.

BIBLIOGRAPHY

1. Lombard: Dublin Journal, September, 1836. (See Am. Jour. Med. Sci., 1835, xv, 320.)
2. Gerhard, W. W.: American Journal Medical Sciences, 1835, xv, 320.
3. Gerhard, W. W.: American Journal Medical Sciences, 1837, xix, 289.

THE TREATMENT OF ARTHRITIS WITH SALTS OF O-IODOXY-BENZOIC ACID*

By JAMES E. COTTRELL, M.D.,
ASSOCIATE PHYSICIAN TO THE HOSPITAL

THE three iodine substitution products of benzoic acid have been known to chemists since 1892. The one with which we are concerned, o-iodoxy-benzoic acid, corresponds to the formula illustrated, containing two double-bonded oxygen atoms linked to the iodine (Fig. 1). When pure, it is a white powder which turns reddish-brown on exposure to light, difficultly soluble in warm water; the dry substance explodes when heated, giving off purple fumes and an odor of iodine. It is a fairly strong acid; its most striking chemical characteristic is its property of acting as an oxidizing agent. The sodium and ammonium salts are readily soluble in water. The iodine content of the ammonium salt is between 40 and 45 per cent.

Loevenhart and Grove,¹ in 1911, described improved methods of preparation of o-iodoxy-benzoic acid, and studied its physiologic action. A summary of their results is as follows. O-iodoxy-benzoic acid is a physiologic oxidizing agent, inasmuch as it can furnish oxygen for the peroxidase reaction of blood. When administered intravenously, it is not hemolytic. It depresses the vasomotor center, causing a fall in blood-pressure; it also depresses the respiratory center and causes a period of apnea in repose, with spontaneous resumption of respiration.

Other rather extensive studies were made about the same time. Arkin² found that sodium o-iodoxy-benzoate exhibits marked bactericidal action, though less effective against staphylococci than against *Bacillus typhosus*, *Bacillus coli*, or *Bacillus pyocyaneus*. He found it to be quite as active in

* Extracted from the American Journal of the Medical Sciences, 1927, clxxiv, 623.

blood-serum as in water. Arkin and Fink³ found it to have no constant effect on the catalase value of blood and tissues. Amberg and Knox⁴ found that the local inflammatory process in an allergic reaction (the reaction to intracutaneous injection of serum in sensitized dogs and rabbits) is diminished by the intravenous injection of sodium o-iodoxy-benzoate, whereas a systemic allergic reaction is unaffected. Hektoen⁵ found that intravenous injection of sodium o-iodoxy-benzoate in dogs stimulates the production of antibodies. Young and Youmans⁶ state that the toxicity of sodium o-iodoxy-benzoate is dependent more on the rate of injection than on the dose administered. All of these investigators agree that the properties of o-iodoxy-benzoic acid are due to the presence of oxygen in the molecule, for o-iod-benzoic acid, which contains no oxygen, has not such properties, whereas they are shared to some extent by o-iodoso-benzoic acid, which contains one oxygen atom less than o-iodoxy-benzoic acid.

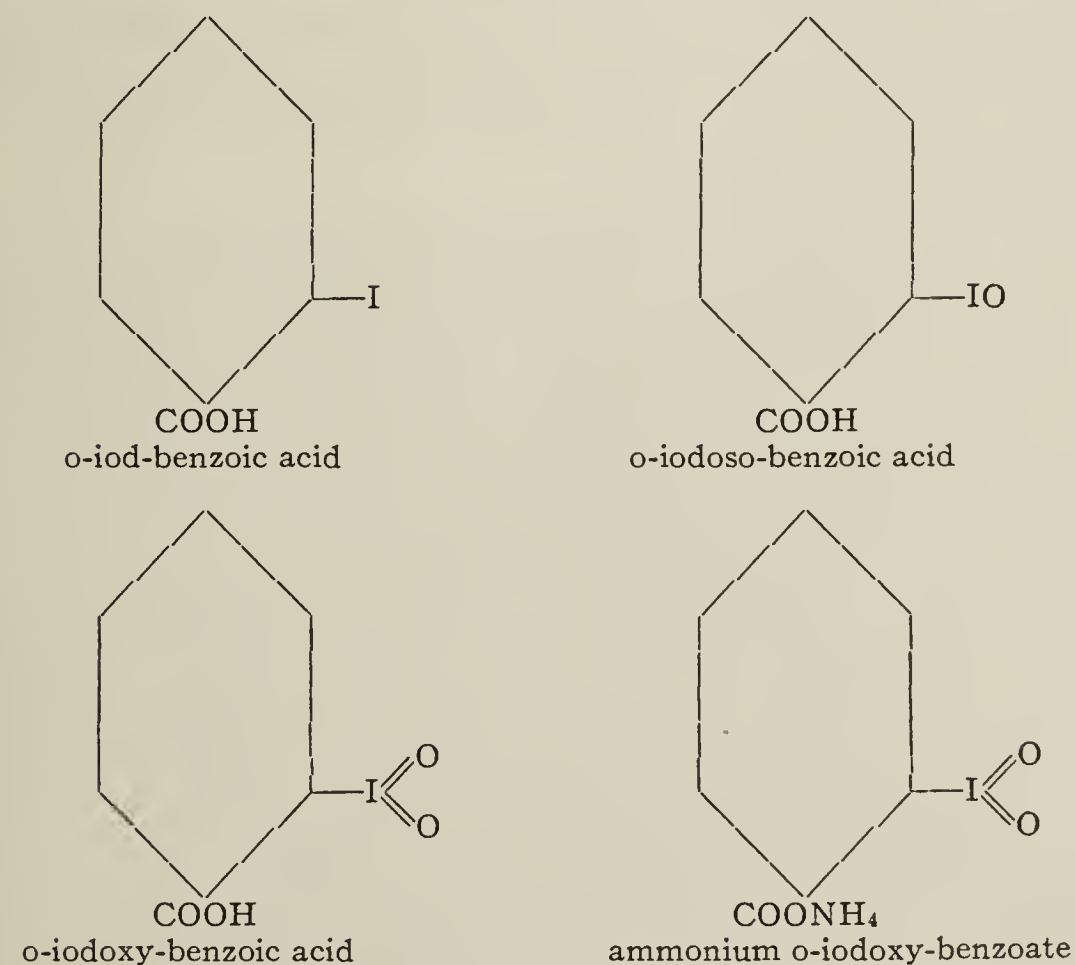


FIG. 1.—Formulæ of iodine substitution-products of benzoic acid.

The systematic use of o-iodoxy-benzoic acid in the treatment of arthritis, under controlled conditions, was initiated by

Young and Youmans^{6, 7} of the University of Michigan. Their series, reported in 1926, comprised 43 patients, ranging in age from the second to the seventh decade; 24 were male and 19 female. All but 4 of their cases were considered to be chronic, and included examples of hypertrophic and atrophic forms, as well as gonococcal arthritis. The duration of the disease in the chronic cases varies from less than a year to more than ten years. Their patients each received from 6 to 25 injections of the drug, in "courses" of about 6 injections, at intervals of three or four days, with a lapse of usually six weeks between courses in those instances where more than one course was given. Of their 43 cases, 56 per cent were said to be markedly improved; 23 per cent moderately improved; 14 per cent slightly improved; and 7 per cent unimproved.

Smith,⁸ of the Boston City Hospital, has reported a series of 33 patients, treated with the drug in a similar manner. Two of his cases were classed as "arthralgia," 12 as acute arthritis, 16 as chronic arthritis, 2 as gonococcal arthritis, and 1 as acute rheumatic fever. All but 3 of his patients showed improvement, as evidenced by diminution of pain or of swelling, or by improvement in function.

The use of o-iodoxy-benzoic acid in the treatment of arthritis, especially infectious arthritis, is based upon its germicidal power for streptococci and staphylococci, and upon its power of stimulating bodily defense mechanisms in general. It seems reasonable to suppose that this non-specific stimulation of reactive processes might also be effective in noninfectious forms, if we agree with many students of the subject that some arthritides are noninfectious. Since Pemberton and his associates⁹ have shown that in many arthritics there is increased oxygen saturation of venous blood and hence presumably decreased removal of oxygen in passage through the capillaries, an obvious possibility is that the power of o-iodoxy-benzoic acid to act as a physiologic oxidizing agent is a factor in the results. A natural explanation for the analgesic effect of the drug, often very striking, may be seen in the similarity of its chemical structure to that of the salicylates, though it is seldom permissible to base deductions as to physiologic action on superficial resemblance in chemical formulæ.

In the spring of 1926, Dr. A. G. Young sent to us, in the Medical Division of the Hospital of the University of Pennsylvania, a supply of the drug, and we have used it in the treatment of a small series of cases, most of them in the University Hospital and the Episcopal Hospital. The series comprises 21 cases. The majority of the patients presented long-standing or very severe arthritis; in almost all, other forms of treatment had been used with little or no improvement. In general, they were cases of such nature as to constitute a severe test of any therapeutic procedure. Seven were male and 14 female. The youngest was twenty-three and the oldest seventy years of age. The proportion of the various types of arthritis, and the average duration in each group, are shown in Table I.

TABLE I.—TYPES OF ARTHRITIS

Type.	Number of cases.	Per cent.	Average duration.
Acute infectious	2	9.5	1.5 mo.
Subacute infectious	3	14.3	1.0 mo.
Chronic infectious (arthritis defor- mans)	13	61.8	4.2 yrs.
Gonococcal	3	14.3	1.0 wk.

In the 13 cases classed as "chronic infectious," the duration of the disease varied from eight months to over ten years, and the age of the patients from twenty-three to seventy years. These patients presented typical chronic arthritis with varying degrees of crippling and deformity, belonging to the group of "proliferative" arthritis, although the history of 1 case suggests somewhat the "menopause arthritis" described by Cecil and his associates.^{10, 11} Several of the patients are still under observation or treatment. Examples of almost every conceivable treatment of arthritis are to be found in the history of the group prior to the administration of o-iodoxy-benzoic acid, including measures for the eradication of infectious foci, injections of foreign protein (Coley's fluid, milk-proteins, bacterial vaccines), physiotherapy, orthopedic treatment, light, radioactive water, and the whole gamut of antiarthritic drugs.

It is not the purpose of this report to enter into a discussion of the classification and etiology of chronic arthritis in general,

but we have considered all these cases as infectious. It seems extremely difficult, in any given case, entirely to eliminate the possibility, or even the probability, of origin from some focus of infection. In all but one of the chronic cases reasonably definite foci were present when the patients came under our care, or they had had operative procedures for the eradication of foci previously. In one acute case, although its infectious nature would be granted by all, no focus was demonstrable. Tuberculous and syphilitic arthritides, and acute rheumatic fever, are not represented in the group. The number of times various common sites of foci were implicated is represented in Table II. The number of instances of tonsillar infection may perhaps be taken with some reservations, but at least in that many cases someone considered the tonsils sufficiently suspicious to recommend their removal.

TABLE II.—INFECTIOUS FOCI

Tonsils	14	} In various combinations (21 patients)
Teeth	7	
Sinuses	3	
Genito-urinary (non-venereal)	3	
Genito-urinary (venereal)	3	
Gall bladder	1	

In all cases, we began by instituting a careful search for foci, investigating routinely the teeth, tonsils, sinuses, ears, the prostate in men, the pelvic organs in women, by clinical, bacteriologic and roentgenologic methods. Where infectious foci were found, they were treated appropriately; and in the earlier cases in the series, time allowed to elapse before beginning treatment with the drug, to evaluate beneficial results which might be obtained merely from the clearing up of focal infection. Following the suggestion of Barrow and Armstrong,¹² the stools of several patients were carefully examined for parasitic protozoa by Dr. Frank B. Lynch, of the Pepper Laboratory of Clinical Medicine, with, however, negative results in each instance. In chronic cases with any impairment of function, physiotherapy was started promptly and continued throughout the course of treatment, consisting of baking, massage, passive movement, and exercise. The usefulness of such measures, properly applied, can scarcely be

overemphasized. Orthopedic measures for the correction of deformities or the relief of pain were used when indicated. In general, other drugs were avoided while using o-iodoxybenzoic acid, except when rarely an analgesic was needed. Salicylates are especially to be avoided, as it is stated that they increase the severity of reactions.

Intravenous injection is by all means the method of choice in administering the drug, and for this the ammonium salt is used. The standard dosage of ammonium o-iodoxybenzoate is 1 gram dissolved in 100 cc. of sterile distilled water, given approximately twice weekly. It is injected by the gravity method, and must be given slowly, allowing ten to twenty minutes for 100 cc. to run in. Rapid administration tends to produce severe reactions. Usually during the injection the patient experiences a curious burning and smarting of the eyes, tongue, mouth, nostrils, and sometimes of the general body surface; this subsides in a few moments. Almost invariably there is a more or less pronounced reaction after the injection; it may come on within half an hour, or it may be postponed for three or four hours. Most often it consists only of slight malaise and perhaps little nausea. Severe reactions are marked by a chill, vomiting, and purging. Very rarely do the patients find the reactions sufficiently uncomfortable to cause them to object to the use of the drug. There is sometimes a rise in temperature of from 1° to 5° F.; more often there is no rise. There seems to be no rule as to the type of cases in which severe reactions occur, and no relation between the degree of the reaction and relief obtained; nor is the reaction at all uniform with successive doses in a given case. In general, more severe reactions were met early in the study of this series, when we were using the ammonium salt extemporaneously prepared by neutralizing a solution or suspension of the free acid with strong ammonium hydroxide; it is to be suspected that chemical impurities aggravated the reactions. Since we have been using the pure, stable ammonium salt, which needs only to be dissolved in the requisite amount of sterile distilled water, the reactions have been far less pronounced. We have never seen clinically the apnea and fall in blood-pressure which are described in laboratory animals.

The mechanism of the reaction is uncertain. Certain features would indicate that it differs from the mechanism of protein shock. There is not the preliminary suppression of polymorphonuclear leukocytes with a secondary rise; in fact, there does not seem to be any constant effect in the leukocyte count, although it is stated¹ that the drug produces a polymorphonuclear leukocytosis. In one patient to whom the drug was administered by mouth a reaction was produced by each dose, less marked than after intravenous injection, but similar in kind. This production of a reaction after absorption from the gastrointestinal tract as well as after parenteral administration argues dissimilarity to the mechanism of protein shock.

For patients with whom great difficulty is experienced in intravenous injection, the drug may be given by mouth or by rectum. For oral administration, calcium o-iodoxy-benzoate is preferable to the ammonium salt, as it seems to produce less gastric irritation. A larger dose, usually 1.5 gram, is given in capsules of 0.5 gram each during a few hours, the patient preferably fasting or eating very lightly before the administration of the drug. The dose is repeated approximately twice a week. Slight nausea is sometimes induced, rarely vomiting, and very rarely (one patient only in this group) any other reaction. For rectal administration the dose is 1 or 1.5 gram of ammonium o-iodoxy-benzoate in 2 per cent solution, given after a cleansing enema; reaction of any kind is unusual, and the drug is usually retained. The therapeutic effect from oral and rectal administration is probably always less marked than from intravenous administration, but the chance of benefit is sufficient to make oral or rectal administration worth while if intravenous injection is impracticable. But it must be emphasized that the intravenous route offers the best chance for satisfactory results, and that oral or rectal administration should be resorted to only if intravenous injection is quite impracticable, or as a supplement to intravenous treatment.

The drug is best given in "courses" of 6 or 8 doses at semi-weekly intervals, with a rest of three to six weeks between courses. One patient in this group has received 3 courses totaling 20 injections. Others have received from 3 to 17

doses. All but 3 have received at least part of their treatment intravenously.

The criteria by which improvement has been judged are diminution in pain and swelling, subjective sense of greater freedom of motion, actual objective improvement in joint function, and increase in the patient's sense of general well-being. Improvement in cases of chronic arthritis is obviously a relative matter; if a patient who has been bedfast is enabled to walk by means of braces and crutches, his state is considerably improved, even though he still be badly crippled. The condition of many of the patients in this group was such that to obtain any improvement at all constituted a therapeutic triumph. All but 3 of 21 patients have shown improvement of varying degree. Relief of pain is often prompt and marked, especially in acute and subacute cases; some patients complain of a preliminary increase in pain for a few hours after an injection. In the great majority of chronic cases with crippling, there has been improvement of function, from slight to very marked. Some patients who were unable to close the hands, or to get the hands to the head, or to rise without assistance, were able, after treatment, to perform those movements. Two patients who had had especially varied and intensive previous treatment both said that this drug was the first thing that had ever benefited them. There are often recurrences of symptoms during the intervals without the drug, but in a case of chronic arthritis the treatment must of necessity be very prolonged. In some cases, after satisfactory improvement at first, we seemed to reach a point after which further improvement did not occur; in at least one the onward progress of the disease seemed not to have been stopped in spite of initial improvement. Table III shows a summary of the therapeutic results in the whole series, and Table IV the therapeutic results in reference to the types of arthritis.

TABLE III.—SUMMARY OF RESULTS

	Number.	Per cent.
Cured	2	9.5
Markedly improved	6	28.6
Moderately improved	4	19.0
Slightly improved	6	28.6
Unimproved	3	14.3

TABLE IV.—RESULTS IN REFERENCE TO TYPES OF ARTHRITIS

Type of arthritis.	Cured.	Markedly improved	Moderately improved	Slightly improved	Unimproved	Total.
Acute infectious	2	2
Subacute infectious .	2	..	1	3
Chronic infectious .	..	4	1	5	3	13
Gonococcal	2	..	1	..	3

We have seen no ill-effects of serious importance. Some of the reactions have been severe, but not alarming; most patients do not even find them seriously annoying. In one case, not included in this series, the reaction to the first injection was severe enough to cause the patient to refuse other treatment. Venous thrombosis occurs at times; its incidence is minimized by careful technic in the injection, and by following the ammonium o-iodoxy-benzoate solution with a little physiologic saline solution to wash out the vein. If some of the o-iodoxy-benzoate solution escapes into the subcutaneous tissues it is only mildly irritant. The fear has been expressed that on account of the high iodine content of ammonium o-iodoxy-benzoate (between 40 and 45 per cent), hyperthyroidism might be induced. But in that combination the iodine is presumably physiologically inert, and the combination is probably not broken down at all in the body. Young¹³ states that practically all of the iodine injected as ammonium o-iodoxy-benzoate can be recovered in the urine still in the same combination; that is, as o-iod-benzoates, or as iodine substitution products of hippuric acid. We have not seen any such case of induced hyperthyroidism.

REPORT OF CASES. CASE I.—M. G., female, aged twenty-seven years (University Hospital). Onset of the disease was about three years previously, with gradual progressive involvement of all the large joints, the fingers, and the spine. She had had tonsillectomy two years, and thyroidectomy one year, before admission. Roentgenogram showed atrophic joint changes. No active foci were found.

The hands could not be clenched. The wrists were limited in flexion and extension. The limit of extension of the elbows was about 165 degrees. There was marked crepitation in both knees,

but passive extension to practically normal range was possible. The hips could be flexed to 90 degrees; the spine was held rigidly.

She received 17 injections in 3 courses. After the first course, she felt greatly improved. She could close both hands, though not tightly; both elbows were extensible to about 170 or 175 degrees, and there was practically no limitation of motion of the knees or hips. She still walked with a limping, shuffling gait.

In the intervals between courses, there was some increase in pain, but no relapse so far as function was concerned. From the second and third courses, she received considerable relief of pain, but little if any further improvement in function. She received physiotherapy throughout the treatment.

CASE II.—Mrs. G., aged seventy years, private patient of Dr. David Riesman. For about five years she had had pain, stiffness, and loss of motion in various joints, most marked in the knees, affecting also the fingers, jaw, wrists, hips and shoulders. The tonsils had been removed; careful search showed no other foci. She had received very extensive and varied treatment.

The knees were swollen. There were atrophy and spindle deformity of the fingers and swelling of the right wrist. Other joints showed no objective changes.

She received 3 courses totaling 20 injections, with a sharp reaction each time. There was marked improvement, especially in the swelling about the knees, and in the function of knees and fingers. She stated that she was better than she had been in years.

CASE III.—A. S. G., female, aged fifty-four years (University Hospital). Onset was three years previously, with swelling and stiffness (at first painless) of the right knee; a year later, the middle finger of the right hand became involved. Two months before admission, the left knee, all joints of both hands, and the left shoulder were affected, with a great deal of pain. She apparently had an attack of cystitis about six weeks before admission. She had been confined to bed for about two months.

There was spindle deformity of all fingers; both wrists were swollen, and flexion and extension limited, but pronation and supination well retained. The elbows were very slightly limited in extension. The left shoulder was extremely painful, and abduction and circumduction almost impossible. There was effusion in both knees. She could not open her mouth fully.

The tonsils were found to be infected, and were removed under local anesthesia; severe shock resulted and almost proved fatal. She received baking and massage, and several colonic irrigations.

There were absolutely no veins available for injection, and she was given the drug by mouth. Really astonishing improvement ensued; after 3 doses she was entirely free from joint pain, said she felt better than she had for ten years, and insisted on going home against advice. The effusion in the knees was less in amount, but still present. Oral administration of the drug was continued at home for a time. At last reports, about three months later, she had maintained her improved condition without recurrence.

CASE IV.—P. C., male, aged twenty-seven years (University Hospital). Duration of the disease was about three and a half years. The left elbow was swollen and tender, and limited to about 150 degrees of extension. At the right shoulder, abduction was limited to about 75 degrees. Both knees were swollen and tender, and limited to about 160 degrees of extension. He was able to walk, but with some pain. The wrists had also been involved.

A tonsillar remnant was removed, and the left maxillary sinus drained surgically. Baking, massage, and ultraviolet radiation were employed. He received 7 intravenous injections of ammonium o-iodoxy-benzoate without any improvement.

CASE V.—N. B., colored, female, aged thirty years (Episcopal Hospital). Onset was a month before admission, with chills, fever, sweats, and generalized joint pain, worst in the right knee and left shoulder.

The tonsils, obviously diseased, and several abscessed teeth were removed. There were some improvement under treatment with salicylates and injections of foreign protein, but joint pain and stiffness persisted. She was given 4 injections of ammonium o-iodoxy-benzoate; pain and stiffness completely disappeared after the first injection. She was discharged as cured.

CASE VI.—R. P., female, aged twenty-five years (Episcopal Hospital). Onset one week before admission, with severe fleeting pains in various joints and temperature of 103.5° F. The following day only the right ankle and the metacarpophalangeal joint of the left thumb were involved; those joints continued to be swollen, red, hot, tender, and extremely painful, disturbing sleep. She had had an appendectomy and oöphorectomy. Frequency of urination and burning were present; she had a profuse vaginal discharge; cervical smears were repeatedly positive for gonococci.

A plaster splint was applied to the ankle, and a gonococcus vaccine administered; the only local treatment of the gonorrhea was by vaginal douches. Some improvement resulted, but she continued to have pain most of the time. She was given 5 injections of ammonium o-iodoxy-benzoate; one of them, by mistake, consisted of 2 grams instead of 1 gram, but there were no untoward results, except some gastric distress. After the first injection, pain in the ankle was markedly decreased, and movement caused very little discomfort; after the second, swelling and redness had subsided, and the splint was removed. After the third injection, gentle massage and passive movement were begun. After the fourth, she was able to be out of bed and had no pain. After the fifth, there was slight return of the pain during one night, but the patient insisted on leaving the hospital against advice.

CONCLUSION. This series is, of course, a small one, and it can only be said that results thus far are encouraging. Only further experience can determine the value of this drug. From present knowledge, we can say that certain cases of arthritis, of various types, are definitely benefited by treatment with the drug; that there are apparently no means of predicting which cases will, and which will not, be benefited; that it seems probable that the drug will prove to have a definite place in our therapeutic armamentarium against arthritis. Surely a drug which has given more than 80 per cent of improvement in various types of arthritis at least deserves a more extensive trial.

A brief scheme for the use of the salts of o-iodoxy-benzoic acid in the treatment of a patient follows:

1. A search for, and treatment of, foci of infection.
2. Administration of ammonium o-iodoxy-benzoate intravenously in doses of 1 gram semiweekly for 6 or 8 doses. Subsequent courses may follow at intervals of three to six weeks, or longer. If intravenous injection is impossible, the drug may be administered by mouth (calcium salt) or by rectum (ammonium salt).
3. Concomitant and persistent use of physiotherapy in chronic cases.
4. Orthopedic treatment of deformities.

Last, the recollection that the drug is only one factor in

the treatment—that these other resources must be used fully, and that in chronic cases the treatment may be persisted in for months or years.

The writer wishes to acknowledge his indebtedness to Dr. A. G. Young of the University of Michigan for many helpful suggestions, and for our original supply of the drug; to Dr. David Riesman, Dr. Joseph Sailer and Dr. Richard A. Kern, each of whom has allowed a private patient to be included in the series.

REFERENCES

1. Loevenhart and Grove: Jour. Pharmacol. and Exper. Therap., 1911-1912, iii, 101.
2. Arkin: Ibid., 1911-1912, iii, 145.
3. Arkin and Fink: Ibid., 1918, xxii, 1915.
4. Amberg and Knox: Ibid., 1911-1912, iii, 223.
5. Hektoen: Proc. Chicago Path. Soc., 1911, viii, 138.
6. Young and Youmans: Jour. Am. Med. Assn., 1926, lxxxvii, 746.
7. Young and Youmans: Jour. Pharmacol. and Exper. Therap., 1926, xxvii, 252.
8. Smith: Boston Med. and Surg. Jour., 1927, cxvii, 305.
9. Pemberton, Hendrix and Crouter: Jour. Metab. Research, 1922, ii, 301.
10. Cecil and Archer: Jour. Am. Med. Assn., 1926, lxxxvii, 741.
11. Cecil and Archer: Am. Jour. Med. Sci., 1927, clxxiii, 258.
12. Barrow and Armstrong: Illinois Med. Jour., 1925, xlvii, 427.
13. Young, A. G.: Personal communication.

SURGERY IN DIABETICS*

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WITH the discovery of insulin by Banting and his coworker Best, the spotlight, so to speak, has been turned on diabetes. Both the medical profession and the general public are becoming more than ever aware of the great amount of study that is being directed toward this persistent disease and its successful treatment. While Banting's name now heads the list of these ardent investigators, too much praise cannot be given the clinicians and chemists in this country as well as abroad, who by their untiring efforts and study have done so much to put the treatment of diabetes on a sound basis, and who paved the way for the final triumph represented by the discovery of insulin.

A result of publicity with regard to this important subject seems to be an increased incidence of diabetes especially in the number of surgical diabetics admitted to the wards of our hospitals. This was most forcibly brought to my attention recently when asked in consultation by my colleague, Dr. John B. Carson, of the medical staff of the Episcopal Hospital. I was amazed, on this visit to his ward, to find not less than 7 cases of gangrene and one of cellulitis associated with diabetes. The prevalence of diabetes is really quite appalling. It has been estimated that from 0.5 to 1 per cent of our population is suffering from this disease, and of this number from 20 to 25 per cent will probably develop some surgical complication. In view of these facts, I believe a discussion of surgery in diabetes to be not untimely.

Surgery always has and probably always will have its hot-house plants. Formerly the prostatic belonged to this class.

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The mortality of prostatectomy in the early history of the operation was a shocking one, due to hemorrhage, uremia, pneumonia, and the like. The imbalance of this type of patient was such that it seemed as though a mere whiff would turn the tide against him. The interest of the profession in hypertrophy of the prostate, stimulated by the brilliant work of Young, Freyer, Deaver and others, has now reduced the operative mortality of this disorder in the hands of the expert to about 5 per cent. It is a justifiable hope that the surgical diabetic will enjoy a similar enviable record in the future, and that diabetics will no longer be the poor surgical risks which they have been in the past. This hope is sustained by the excellent results obtained since the introduction of insulin, together with metabolic and dietetic study and control of the diabetic patient, so that the time is near at hand, I hope, when an operation on such a patient will be undertaken with the same degree of confidence as on the nondiabetic. But this can only be brought about by coöperation between the surgeon and the medical man. To my mind it is essential that the surgeon attend to the surgical condition and his medical colleague to the diabetes. I believe the medical man needs this note of warning more than the surgeon. In my service at the Episcopal Hospital I frequently send an operated diabetic case back to the medical ward, where the doctors and nurses who have had the patient in charge before operation and are familiar with every aspect of the case, can continue the treatment. If the case is sent to the surgical ward, the medical staff is frequently called in consultation.

Surgical diabetes readily falls into two main types: first, diabetes complicated by a surgical disorder, such as gangrene, carbuncle, etc., serious and often emergency conditions; second, a surgical disease associated with diabetes, such as gall-stones, carcinoma, prostatic or uterine trouble—conditions which may be just as serious as far as the patient is concerned, but which do not require such hasty and prompt intervention as the first group.

The surgical diabetic will probably always be more of an operative risk than the nondiabetic on account of the unfavorable factors of age, lowered resistance to infection and the

danger of coma. It was formerly thought that one of the greatest dangers in operating on a diabetic was that of infection, but with modern precautionary methods and technic, a clean wound in a diabetic should heal almost as promptly as in a nondiabetic. Coma, at one time the most dreaded post-operative complication, was usually found to be due to one or another cause, such as preoperative starving, ether or chloroform anesthesia, postoperative starvation and withholding of fluids, and delayed operation in infected cases. But today prompt intervention is considered imperative in infected cases. They are as much emergency cases as any that I know of in surgery, except hemorrhage. The relief of sepsis means relief of acidosis. I am in entire accord with Joslin when he says: "The patient should be treated for the operation rather than the diabetes. Get him successfully through the operation first, and then if you like, treat him for the diabetes the rest of his life. If you treat him first for the diabetes and second for the operation, the duration of that case of diabetes is apt to be brief."

Where there is no need for haste in surgical diabetes the regulation of the diet is of great importance. An adult, during activity, requires a diet yielding 30 calories and during rest 25 calories of energy in twenty-four hours for every kilogram of body weight. In uncomplicated cases the total calories required should be determined by some accepted chart, such as the Du Bois tables, or by multiplying the ideal weight in kilograms by 30 or 25 (as the case may be). In arranging the proportion of carbohydrate, protein and fat, it must be borne in mind that fat is oxidized only in a definite mathematical proportion to the amount of glucose oxidized. This is known as the ketogenic or antiketogenic ratio; that is to say, there must be a definite proportion between the substances that form glucose in the body and those that form fatty acids. A simple and satisfactory plan is to arrange a diet with three times more fat than carbohydrate, since the relative oxidation of these two substances is in about that proportion. One gram of protein per kilogram of body weight should be allowed for adults, while in children the proportion is about one and a half to each kilogram.

The dictum that there are no hard and fast rules in surgery applies with greater than ordinary emphasis to surgical diabetes. Each case presents problems of its own, particularly in the presence of infection or gangrene. In the uncomplicated case the administration of insulin usually produces a rapid fall in the blood-sugar, but this does not happen so regularly in the complicated ones. In some instances only the slightest change is noted even after large doses. Where there is suppuration and fever the reduction in blood-sugar is notably slight and slow. I believe such cases should be put on one-half or even one-third the usual diet in order better to control the hyperglycemia and of course the glycosuria. Moreover, it often is useless to attempt to put these cases on a full maintenance diet because they will not eat it. Even on a restricted diet large doses of insulin may fail materially to reduce the blood-sugar. The probable reason why these patients require so much insulin is likely due to the fact that the diseased pancreas is so overwhelmed by the toxemia that its function is seriously impaired. But as the toxemia lessens the amount of food can be increased and the dose of insulin necessary to control the blood-sugar often becomes smaller. This all points to a return to a more normal pancreatic function, and eventually in many cases dietary restrictions without the aid of insulin may suffice. In emergencies there is no fixed plan of treatment other than that suggested by the frequent determination of the blood-sugar and the degree of acidosis. In the chronic case where haste is not indicated and the patient can be prepared for operation at leisure, regulation of diet, either with or without very small doses of insulin, will be all that is required. It is of advantage to feed these cases right up to the time of operation, thus storing up sufficient glycogen and reducing the danger of acidosis to a minimum. Carbohydrates are also useful in avoiding acidosis since their oxidation is essential for the combustion of fat. Postoperative acidosis may also be minimized by the preoperative administration of 20 to 50 grams of glucose intravenously, followed by an appropriate dose of insulin subcutaneously. Fluids should be liberally given by mouth up to the time of operation, and in severe cases should also be supplied by

enteroclysis or hypodermoclysis, and even intravenous infusions may be of great value.

In the presence of infection it is often very difficult to get the patient sugar-free before operation. While it can be done by administering large doses of insulin, this may be unsafe and impractical, and personally I believe it is unnecessary.

The danger of postoperative alkalosis, though a rare condition, should always be thought of. It is usually due to the preoperative administration of alkalies, and sometimes to prolonged and excessive vomiting, which causes the patient to become depleted of free hydrochloric acid. Tetany is the most important and constant clinical symptom of alkalosis.

The danger of coma should also be continually in the mind of the attending physician and nurses. It may develop very insidiously or may come on suddenly like a summer storm. Vomiting, nausea, restlessness, fatigue, dizziness, dry tongue and air-hunger, characterized by deep and rapid breathing, are some of the signs and symptoms of acidosis and impending coma.

In operating on a diabetic the anesthetic is most important. Neither chloroform nor ether should ever be given. Not only do they require preoperative starving, but they are too often followed by nausea and vomiting, and produce a hyperglycemia, while during ether anesthesia the formation of glycogen is said to cease. Personally, my first choice is spinal anesthesia, second, gas-oxygen, and third, local anesthesia. I am not in favor of the last-named, because I believe the injection of the anesthetic traumatizes the tissues and favors infection and sloughing.

Carbuncles represent probably the most serious surgical complication of diabetes and hitherto have yielded a very high mortality. At present, however, the prognosis is more favorable, depending, of course, on the degree of diabetes and acidosis, the age of the patient, the size of the carbuncle and the resulting sepsis. As a rule, these cases are emergency ones and require prompt radical treatment. The severe cases should be treated as outlined above with glucose and insulin, and operation should consist of complete excision of the carbuncle under gas anesthesia. The less severe cases may be

treated dietetically and with local applications of magnesium sulphate paste, or by crucial incision and hot packs.

A less serious, perhaps, but certainly more common complication is gangrene, usually of the lower extremities. It seems to affect men past middle life more often than women. Most of the patients are in the sixth decade, although I have operated upon a patient only thirty-five years of age. There is usually marked arteriosclerosis of the endarteritis type, and as in senile gangrene, the chief predisposing factors are uncleanliness and trauma. The prodromal symptoms are pain, numbness and coldness. The gangrene is usually of the dry type, involving the toes, although the foot and leg may be affected, but these more frequently develop moist gangrene. In either type examination oftentimes fails to disclose pulsation at the dorsalis pedis or posterior tibial artery, and frequently pulsation is also absent at the popliteal or the femoral artery in Scarpa's triangle. When these cases come to amputation, as they often do, the artery will usually be found to be thrombosed throughout its extent. Dry gangrene without infection, and when the patient is in good condition, can be prepared leisurely for operation and brought as near as possible to a normal sugar balance. In my early experience with diabetic gangrene I was inclined to believe the gangrene to be due to some direct action of the sugar or its derivatives—in other words, that it was probably a chemical process. If this were true, then insulin and diet regulation should be of great value; but I am now absolutely convinced that in the majority of cases the gangrene is due to an insufficient blood supply. Nearly always there will be found not only endarteritis, but also thrombosis, and frequently back of these a history of trauma. Many of the cases which develop pain, numbness or coldness of the toes or feet, or even marked cyanosis or lividity, clear up after a few days' rest in bed, together with the application of dry heat, diet and the administration of insulin. Buerger and Bernheim have each devised methods for increasing the blood supply to the affected part in these cases, with reports of good results.

I believe that practically all of these cases are thrombo-endarteritis obliterans and that the establishment of a good collateral circulation may save the tissues. In any case, if

gangrene develops, that part is dead, never to be revived, no matter what type of treatment is used, and amputation is imperative. Occasionally, however, one sees a case of superficial gangrene of the dorsum of the foot in which only the skin is involved, due to the plugging of some small artery, and with the separation of the slough, healthy granulation will be found underneath and healing will take place. Cases of cellulitis require early free incision and drainage.

Moist gangrene, as a rule requires immediate surgical attention. The only exception to this rule is where favorable conditions may permit delay, as when the patient is robust and not suffering from marked cardiorenal disease, and is not especially toxic. The roentgenogram is of aid in demonstrating the presence of bone changes, or marked arteriosclerosis of the tibial arteries which usually develops in such cases.

The diabetic with gangrene, moist or dry, usually will sooner or later require amputation. If amputation is necessary the preoperative treatment, anesthesia, and postoperative treatment should be along the lines already indicated. At operation no tourniquet or constricting band should be applied. The tissues should be handled as delicately as possible. Drainage is not always necessary, but if used should consist of gauze or rubber tissue. Drainage tubes are harmful because their pressure may be sufficient to produce sloughing of the tissues. Conservative operations or those planned to save tissue should be avoided. The site of amputation will depend on the extent of the gangrene. When it involves the toes and a portion of the foot, amputation should be done at the upper third of the leg; when it extends well above the ankle, the point of amputation should be above the knee. The fact that no pulsation is palpable at the femoral artery beneath Poupart's ligament, does not indicate amputation through the thigh, inasmuch as frequently the point of choice is below the knee, the stump being well taken care of through the deep femoral and collateral circulation. There is also less shock at this point, and furthermore the stump is more suitable for an artificial leg.

Dry gangrene of the toes is often difficult to handle, for after the removal of one or more toes the wound often fails to heal and a new rim of gangrene appears around it, so that

another operation is necessary. This, of course, is not always the case and the conservative measure of amputating the toe or toes should be tried first. It is my experience that amputation of the great toe or the small toe gives better results than removing the middle ones. Foot amputation, such as Chopart's or Lisfranc's, should be avoided, for these cases usually turn out badly and one finally ends by taking off the leg. Periarterial sympathectomy, I believe, is absolutely useless in diabetic gangrene. One need only dissect out the artery of an amputated leg to see why no results can be expected. The artery is entirely thrombosed.

In not a few instances the onset of gangrene brings the patient to his doctor, and it is only then he first finds out that he has diabetes. The gangrene may, therefore, be a blessing in disguise, for very often in such instances and in cases of long-standing diabetes, amputation seems to be very beneficial. The patients improve, put on flesh, and keep well on a dietary régime alone, without any medication.

In noninfected surgical cases accompanied by diabetes, where the cardiorenal system is not seriously damaged and haste is not imperative, so that the patient can be leisurely prepared for operation, there is an excellent chance of a very successful operation. However, the infected surgical conditions complicated by diabetes, a good example of which is acute suppurative appendicitis, are usually very serious. Their salvation is early diagnosis and operation while the lesion is still confined to the appendix. Once peritonitis develops, the prognosis is most grave. For a diffuse peritonitis the Murphy-Fowler-Ochsner treatment should be employed with the idea of localizing the infection. As nothing can be given by mouth, glucose or salt solution should be given by enteroclysis, hypodermoclysis or intravenously. In these cases one is, so to say, between the devil and the deep blue sea. To starve a patient favors acidosis and coma, and to feed him increases the peristalsis and leads to a dissemination of the peritonitis. Codein should be given freely. I believe that where the patient exhibits good resistance and is seen in the early stages of peritonitis, immediate operation is advisable. In appendiceal abscess, incision and drainage is the indicated procedure. No attempt should be made to remove the appen-

dix, unless it is easily found, for in searching for the appendix, the necessary breaking up of adhesions and of the lymph barriers will oftentimes open up new avenues of infection which may be the patient's undoing.

Acute cholecystitis, other than the perforative type, which fortunately is very rare, as a rule does not demand an emergency operation. Such patients, therefore, can often be successfully carried along until the infection subsides, and an interval operation can then be considered. Should operation be decided upon either during an attack or in the quiescent stage, cholecystostomy is the procedure of choice, since it requires less time and less profound anesthesia than cholecystectomy.

The routine examination of all obese patients admitted to the surgical ward of a hospital, in addition to the usual urine and blood analysis and physical examination, should consist also of a blood-sugar estimation. I have had the experience, and no doubt others have also, that often a diabetic patient will run a sugar-free urine, operation will be undertaken and the patient go into postoperative coma. A blood-sugar determination in such cases would have avoided this unpleasant catastrophe.

There is no need for the present appalling mortality of the surgical diabetic. The trouble is that these cases are rushed into a hospital at the last minute in a toxic acidotic state, usually with or in impending coma, and the result too often is death. The hope of the surgical diabetic lies in the hands of the family physician, the diabetic specialist and the patient himself.

In addition to the scientific treatment of the diabetes, the patient must be imbued with the necessity of avoiding trauma and uncleanness, and if a surgical condition should arise, he should come to the surgeon in good physical shape at the incipient stage of the complication with a controlled blood-sugar, a rested pancreas and free from acidosis. When this happy combination takes place, then indeed will surgical diabetes have lost many of its terrors, and will enjoy the same margin of safety as attends other operative procedures, such as prostatectomy referred to at the beginning of this paper.

POLYCYTHEMIA (ERYTHREMIA) AND ITS NEWER TREATMENT

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MIDWAY to the birthday anniversary this hospital is now celebrating, a new clinical entity was described which has provoked much contemplative comment ever since. Within a decade, Vaquez and Osler recorded middle-aged patients with florid faces, fiery mucous membranes, enlarged spleens and erythrocyte counts well over the usual 5,000,000 per c.mm. Osler wrote in English, wrote more persistently, named the disease polycythemia vera, and in return most American and English texts now call it simply Osler's disease. A little later, Gaisbock noted the same syndrome occurring in the fifth and sixth decades, but in addition an elevated blood-pressure, and called the combination polycythemia hypertonica.

When confronted with a high erythrocyte count the diagnosis of Osler's disease is not clinched by any means, for polycythemia may be a symptomatic expression of some other underlying serious malady. To clarify the confusion arising here, Parkes-Weber has proposed the term *erythremia* to be applied exclusively to Osler's disease, and he classifies all secondary polycythemias under the term *erythrocytosis*. Obviously, such a conception is borrowed from the accepted classification of leukocyte pathology and is meant to convey much the same idea of causation to the medical mind.

The factors which will reflect themselves in an erythrocytosis, may be grouped under the general heads of *mechanical* and *infectious*.

The mechanical group includes any disease which will produce long-standing tissue anoxemia, stimulating the bone-marrow to an overproduction of erythrocytes as compensatory oxygen carriers. People living at high altitudes show this

blood change so constantly that it has been considered physiological. More rarely we see erythrocytosis as a symptom in the following pathological conditions:

Congenital stenosis of the pulmonary valve.

Mitral stenosis.

Emphysema.

Asthma.

Chronic uremia.

The infectious group is not so clearly defined, but there seem to be a number of patients suffering from long-standing suppurations, especially those with renal suppuration, who later develop erythrocytosis.

As an example, I may cite the case of a man, aged fifty-eight years, sent to me through the courtesy of Dr. Louis Wright, of Harrisburg, in the summer of 1927. For many years previous he had pyuria, and combined examinations with the cystoscope and pyelogram had shown one kidney destroyed until it was practically only a pus sac. Headaches and a florid face supervened. His erythrocyte count was found to be 10,000,000. In the absence of any history of red faces in the family, I felt justified, in view of the history sequence, in considering this a case of erythrocytosis secondary to chronic renal suppuration.

We have had no cases of erythremia on the medical services since my appointment to the staff of this hospital; in fact as far as can be determined from the records, there have only been 2 reported cases in the history of the hospital. One of these was a private patient of Dr. John B. Carson's, the other was a patient on the service of Dr. A. A. Stevens, staying in the ward a very short time in February, 1917.

The latter patient was a man, aged forty-one years, born in Austria, who emigrated to America five years before his hospitalization. He was a married laborer with five children, had never been ill, but had always used alcohol and tobacco to excess. His only complaints were a loss of appetite, a feeling of weakness and an increasing discomfort in the epigastrium, which had begun two months previously. His face and mucous membranes were flushed (almost cyanosed), he had a coated tongue and poor teeth. The heart and lungs were normal, the blood-pressure was 105/75. The liver was

palapable, 6 cm. below the costal margin, and had a granular edge, though it was not tender. The urine showed a light cloud of albumin and a few leukocytes. The Wassermann reaction was not recorded.

The blood-counts were as follows:

Hb.	R.B.C.	W.B.C.	Polys.
120	6,500,000	6,880	90 per cent
120	7,740,000		

This is evidently a case of erythremia with a probable Laennec's cirrhosis of the liver.

The rôle of alcohol and lues in the etiology of this disease has been proved to be *nil*. In a series of 8 cases, which I have had the good fortune to observe, these factors played no part. They may accompany the disease, but are not its cause.

The true etiology of erythremia is unknown. The accepted theory at present postulates a factor which stimulates the erythrocytic portions of the long-bone marrow to unusual activity. The evidences of this activity are the increased cell-count and the appearance of normoblasts and megaloblasts in the blood smear. A further evidence is seen in those rare instances in which erythremia becomes pernicious anemia, showing an exhaustion of the erythropoietic faculty of the marrow. However, I do not believe that enough attention has been paid to the reverse of the medal. It is plausible to conceive erythremia as a failure of the blood-destroying mechanism, and, having lost this function, the individual must carry in his blood-stream an abnormal number of red blood cells which are not vital, so cannot carry oxygen. The need for oxygen carriers stimulates the bone-marrow to the further production of erythrocytes to replace those which, though dead, cannot be destroyed. The only reference to any consideration of this phase of the question is the notation by Dietl that he considers presclerotic changes in the spleen (the major blood-destroying organ) of etiologic importance in polycythemia.

The clinical recognition of erythremia is often accidental, the discovery being made during a routine blood-count, or as a result of one of the complicating accidents of the disease which are usually startling enough to fix attention on the underlying cause. An apparently well individual may have sudden

epistaxis, hemoptysis, hematemesis or metrorrhagia which proves intractable and may threaten life, after which the erythremia is discovered. A physician under my observation had never been in bed in his life until, at the age of sixty-six years, he bled profusely from the cavity of an extracted tooth. The bleeding was stopped after forty-eight anxious hours, and his erythrocytes were later found to be 6,000,000. Paralysis from cerebral thromboses, or severe pain anywhere, due to peripheral thromboses, may be the first intimation of the trouble. An associated arteriosclerosis together with the increased viscosity of the blood may prove too much for the heart, and the patient may first be seen in the midst of a cardiac decompensation. In contrast to this is the type of erythremia known as Ayerza's disease, in which cardiac decompensation is simulated and the patient turns almost black for a short period of time, though no cardiac lesion can be demonstrated. Ayerza considers this group due to luetic sclerosis of the pulmonary artery.

The less-outstanding symptoms of erythremia would certainly not lead to its diagnosis, for they are due in the most part to passive congestion and increase in the blood-volume. Headache, usually of the migraine type, has been the most common single symptom in my experience. The most interesting cerebral symptom I have noted occurred in a middle-aged peripatetic furniture polisher. While plying his trade on a very hot day he suddenly became so dizzy that he fell to the ground and vomited, though he did not lose consciousness—evidently the syndrome of Ménière. At the time he was seen, and the diagnosis of erythremia established, he was perfectly rational; but a year later he developed a psychosis and has been confined to an institution. Jagic and Spengler consider such personality changes due to cerebral thrombosis and note that the psychosis may precede the appearance of the erythremia. Fatigue and drowsiness, or at times sleeplessness, are frequent complaints. One patient complained of dull aching of the back and legs on arising in the morning, promptly relieved by his usual hot bath. The venous circulation in the legs was unimpaired. Rarely, a patient will notice an enlarging abdomen.

On physical examination the patient has a flushed face with dilatation of the venules of the nose and lobes of the ears. The veins are overfull and the scleral vessels markedly injected. The lips are purplish, the tongue and mucous membranes of the mouth a fiery red. If the process has been of any duration, the temporal arteries will be serpentine. The heart and lungs are usually unaffected. In a majority of cases the spleen is palpably enlarged, though its size varies so notably even in untreated patients that this is not in any way a constant finding. In one case, however, the rise in erythrocytes was not noted until several months after a very large spleen was found. Individuals in the fifth and sixth decades have often an associated hypertension, varying from 180 mm. upward. Gaisbock classified them as a separate group—polycythemia hypertonica—but most other authors consider the hypertension due to renal sclerosis. At earlier age levels the blood-pressure is normal.

Confirmation of the diagnosis rests on the blood-count. The hemoglobin value averages 115 to 130 per cent. The erythrocytes number between 6,000,000 and 8,000,000 per c.mm. The highest hemoglobin value reported was 178 per cent, and the highest erythrocyte count 14,000,000. The erythrocytes are normal in size, shape and staining qualities. Often there are normoblasts and megaloblasts seen. There is always an associated leukocytosis ranging between 10,000 and 12,000 per c.mm. with a polymorphonuclear preponderance. In certain blood smears a few myelocytes may appear, but they are merely evidences of marrow stimulation and should arouse no thought of leukemia. The blood platelets are not increased, the clotting time is normal or even reduced, the bleeding time is a little lengthened. The blood-volume is increased in direct proportion to the increase in the red cells, and during effective treatment is reduced proportionately.

Erythrocytosis and erythremia are chronic, and of themselves harmless, their menace to comfort or life resting in the complications which may occur during their course. Some of these have already been mentioned as appearing in the rôle of presenting symptoms. Gout is a rare complication, but deserves a little attention. Recently Davis has reported such

a case, with painful feet and hands, a blood-uric acid of 4.7 mg. and gouty deposits shown in the bones of the feet by *x*-ray. The physician of whom I spoke above had a more striking attack of the same nature. In anticipation of a European trip he was in the midst of a course of prophylactic antityphoid vaccine, when two days after the second injection he had excruciating pain in the feet, with redness, swelling and hyperesthesia of both first metatarsal joints. The pain lasted several days, yielding only to large doses of opiates. He had to postpone his trip two months. During the acute stage his blood-uric acid was found to be 24 mg. per 100 cc., and his creatinine 7 mg. Doubting such incredibly high values, I requested a sample of the blood sent to Philadelphia, where it was examined in the laboratory of the Philadelphia General Hospital, and the uric acid found to be 23 mg. with a creatinine of 6.3 mg. Since then his uric acid has ranged between 8 and 11 mg. and the creatinine has fallen to 2 mg. As an interesting corollary to this attack, two of his three brothers have been found to have high blood-uric acid values.

Death in erythremia is due to intercurrent disease in most instances, but there are some who succumb to cerebral hemorrhage, thrombosis or embolism, or during uncontrollable hemorrhage from any of the mucous membranes.

Certain general principles must guide the treatment of these patients. Bodily and mental rest must be strictly enforced to avoid cerebral accidents. Fluids should be prescribed to overcome the tendency to high blood viscosity, and yet no drug can be used which will raise the blood-pressure, especially not coffee or alcohol.

The necessity for reducing the number of erythrocytes and the blood-volume has engaged every author since the first description of the disease, venesection being, of course, the oldest method. It is best to reserve this for any serious complication or crisis, for the corpuscles regenerate very rapidly and the improvement is only temporary. Radiation of the long bones, large doses of arsenic and the use of benzol meet with the same objection, and, in addition, have a factor of danger in that they cannot be accurately controlled and an aplastic anemia may result. All these difficulties have been

fairly well overcome since 1918 by the introduction of phenylhydrazine hydrochloride into the therapy of erythremia and erythrocytosis.

Since Hoppe-Seyler first used it in 1885, phenylhydrazine hydrochloride has been known as a powerful erythrocytic poison. In 1918 Eppinger and Kloss first used it hypodermatically in erythremia with good therapeutic results, but the injections were so irritating that Owen, in 1924, tried oral administration with splendid success. At present, treatment is begun giving 0.1 gm. of the drug daily in a capsule, making a daily blood-count to follow its effect. An *increase* in the leukocytosis is the first sign of drug activity, followed in a few days by a fall in erythrocytes, which will be quite rapid. Because of its profound cytolyzing power, phenylhydrazine must be stopped while the erythrocyte count is still high, for at least a million cells per unit volume more will be destroyed before the effect has worn off. Failure to comply with this plan nearly proved disastrous to one patient, for he became jaundiced from the too rapid release of pigment into the blood-stream and suffered a severe anemia. Fortunately, his count did not fall below 2,500,000 and regeneration began soon again. The total initial dose should not exceed 3.5 gm., and subsequently the patient can keep his blood very well under control by the use of 0.1 to 0.3 gm. *weekly*. There are no toxic effects. One might suppose that the drug or the cytolysis should damage the liver or kidneys. No such clinical effect has been reported, and, after feeding overwhelming doses of the drug to dogs, Allen and Giffen found no evidences of impaired renal or hepatic function. Extra caution must be used in the drug treatment of advanced cases, or in those over sixty years of age, as Giffen and Conner of the Mayo staff have recently shown. They cite four instances of untoward effects in these individuals, and their summary is worth quoting:

“1. Patients with advanced polycythemia vera of a grade necessitating confinement to bed should not receive phenylhydrazine.

2. Extreme caution should be observed in administering phenylhydrazine to patients more than sixty years of age, to patients who

have marked arteriosclerosis and to patients who manifest evidence of advanced visceral injury. It is wise to give a very small dose to such patients, possibly only 0.1 or 0.2 gm., and to observe the effect over several succeeding days. This, however, should not be done if the patients are bedridden.

3. Patients who have probably had thrombosis should be treated cautiously.

4. Every effort should be made to keep the already sluggish circulation as free as possible. Treatment is best carried out with the patient ambulatory. If a patient is under observation in a hospital, he should be kept on his feet as much as possible; massage and exercise in bed have been proved to be satisfactory measures."

The contemplation of erythrocytosis and erythremia should give us particular satisfaction. We have advanced beyond Osler and Vaquez. We recognize the disease more frequently clinically, and bring more complete laboratory studies to the support of our diagnosis. Far more important has been our therapeutic advance, for we now have in our hands phenylhydrazine hydrochloride which, judiciously used, will keep these patients comfortable and active over an indefinite period.

BIBLIOGRAPHY

1. Osler: Trans. Assn. Am. Phys., 1905, xviii, 299.
2. Vaquez: Bull. Med., Paris, 1892, vi, 849.
3. Gaisbock: Quoted in Jagic and Spengler.
4. Parkes-Weber: London, 1921.
5. Dietl: Quoted in Jagic and Spengler.
6. Ayerza: Cardiacos nigros, Clinical Lecture, Univ. of Buenos Aires, 1901.
7. Jagic and Spengler: Klinik und therapie der Blutkrankheiten, Vienna, 1928, p. 235.
8. Davis: Jour. Am. Med. Assn., 1929, xcii, 1595.
9. Hoppe-Seyler: Ztschr. Biol. Physiol. Chem., 1884, v, 11, 34.
10. Eppinger and Kloss: Therap. Monatschr., 1918, xxxii, 322.
11. Owen: Bulletin Johns Hopkins Hospital, 1924, xxxv, 258.
12. Allen and Giffen: Ann. Int. Med., 1928, i, 655 and 679.
13. Giffen and Conner: Jour. Am. Med. Assn., 1929, xcii, 1505.

DIFFERENTIAL DIAGNOSIS OF CHRONIC INFLAMMATORY LESIONS OF LUNGS AND MINIMAL APICAL TUBERCULOSIS

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OF the patients examined in the Chest Clinic of the city of Philadelphia located at the Episcopal Hospital, a few have been proved to have other than tuberculous lesions of the lung. It is usually only after a most exhaustive study that a definite differential diagnosis can be made between a chronic nontuberculous infection and incipient or minimal tuberculosis. The following data have been collected in a review of the total number of new cases admitted to the Clinic during the first eleven months of 1929: there were 465 new cases examined; in 352 of these no disease of the lungs could be found, while in 113 pulmonary disease was present; of these 104 cases (92 per cent) were diagnosed tuberculosis, while 9 cases (8 per cent of those with pulmonary disease and 1.9 per cent of all new cases examined) showed chronic nontuberculous pulmonary inflammation.

On examination of the chest the following physical signs may be found in patients with either tuberculosis or nontuberculous chronic inflammation:

1. Restricted expansion of the upper half of the chest.
2. Diminished expansion of one side of the upper chest.
3. Poor nutrition of the muscles of the upper chest.
4. Roughened breath sounds.

In a low-grade tuberculous infection we are apt to have, in addition, an occasional fine crackling râle present, either following expiratory cough or with quiet breathing. When râles are present and constant they are extremely important as a diagnostic sign of a tuberculous lesion. They may be present but are very infrequent with a chronic nontuberculous inflam-

mation, and frequently are absent in genuine minimal apical tuberculosis.

The symptom-complex of a chronic nontuberculous apical lesion may also be almost identical with that of an early tuberculous focus. This consists of:

1. Fatigue.
2. Malnutrition, or recent loss of weight.
3. Frequent colds.
4. Persistent cough.
5. Expectoration.
6. Slight fever.

The history of the case usually points quite definitely to one or the other of the conditions, and therefore should be taken with great care and given full consideration in the making of a differential diagnosis. The items that almost invariably indicate a tuberculous lesion are:

1. Pleurisy with effusion.
2. Small or marked hemoptysis.
3. Fistula-in-ano.
4. Contact with a sputum-positive case of tuberculosis.

The laboratory differentiation is also very helpful and should always include the following:

1. Tuberculin diagnostic test.
2. Analysis of sputum whenever there is expectoration.
3. Sinus transillumination or x-ray.
4. Lung x-ray.

The intradermal tuberculin test indicates whether or not a tuberculous infection is present. It should be done on every lung patient. Its importance as a diagnostic test in children is fully appreciated by the profession. In adults, also, a negative tuberculin reaction, although rare, practically rules out the possibility of the presence of a tuberculous infection.

While it has not yet been definitely proved, it may be suggested that the intensity of the tuberculin reaction is of some help in making a decision regarding the diagnosis. The stronger the reaction with the highest dilution of Koch's old tuberculin, the greater is the probability of the presence of tuberculosis in the so-called borderline cases.

If there is sputum it should be examined repeatedly for

tubercle bacilli; but it must be remembered that a negative sputum does not prove that the lung lesion is other than tuberculous. In incipient or minimal tuberculosis the lesion is not sufficiently advanced to throw out tubercle bacilli in the sputum.

X-ray examination of the sinuses will show whether or not there is a focus of infection here that may be responsible for the inflammation of the lung apices. X-ray examination of the lungs demonstrates the presence of a lesion and shows its anatomical distribution and extent, but does not aid in any great degree in differentiating between a tuberculous focus and a lesion of nontuberculous origin in the apex.

The tonsils should be examined in every case by a competent otolaryngologist, and his opinion obtained as to the degree of the disease in the throat. However, a subacute or chronic infection of the tonsils may exist along with a tuberculous lesion of the lung and not be responsible at all for the pathological state of the lung tissue.

Any patient in poor general health with the symptoms given above, and having a low-grade sinus infection accompanied by a lesion in the lung, whether this be tuberculous or not, is definitely in need of the so-called "tuberculosis cure" which includes rest in bed, a high-caloric diet and abundant milk. The amount of rest required should be proportionate to the severity of the symptoms and is the most important and effective single item of treatment. Every case of lung infection should be given from four to six weeks of rest and full diet; for with such care a large majority of the cases will show a marked improvement with no other treatment than symptomatic medication. Autogenous vaccines may be tried with caution in the nontuberculous cases. This rest, diet and vaccine treatment should always be given adequate trial before more radical surgical measures are adopted. The removal of the tonsils and the drainage of the sinuses should be dependent chiefly on the definite evidence of pus in these foci, the presence of a damaged heart valve, kidney infection or inflammation of the joints.

A chronic inflammatory process may occur in the bases of the lungs or in the midportion; but here it offers little difficulty

in differential diagnosis, for the usual minimal tuberculous process begins in the lung apices in adults.

CONCLUSIONS. The similarity of the symptoms and physical signs found in cases of mild chronic inflammatory lesions in the lung apices with those of minimal tuberculosis makes the difficulty of differential diagnosis obvious. However, if the patient gives a history of contact with open tuberculosis, or of hemorrhage, pleurisy or fistula-in-ano, we may safely consider the lesion to be tuberculous, especially if there is a strong tuberculin reaction. On the other hand, sinus and tonsil examination may definitely indicate these foci as responsible for the pathological state of the lung apices.

EMPYEMA IN CHILDREN*

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IN a consideration of empyema in children the cases may be classified into two groups: those that occur during infancy between the first and third years, and those that occur after the fourth year of life. In the series herewith reported, the mortality percentage for the first group was 56, 24 and 17 per cent respectively in the first, second and third years of life; while in the second group the mortality was only 11 per cent.

The high death-rate in early infancy is probably due to several factors. The pneumonia, which is usually the forerunner of an empyema, is often of the bronchial type and frequently follows some debilitating disease, so that when the empyema develops, the child's resistance is at a low ebb. Generally, also, the pneumonia is of the lobar type, and is not localized to one area but several patches may exist simultaneously, producing a severe toxemia and thus reducing the infant's reserve to a minimum.

Other factors involved are that in infants the empyema is apt to develop at an earlier stage in the pneumonia than it does in older children, that is, before resolution has taken place; furthermore, infants seem more prone than older children to develop a septicemia associated with the pneumonia; and finally, there is no doubt that during early infancy the pneumonia is more apt to be complicated by such serious conditions as meningitis, peritonitis, pericarditis and otitis media.

This study is based upon 291 cases of empyema observed in

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our services at the following hospitals: Mary J. Drexel Home, 103 cases; St. Christopher's Hospital for Children, 101 cases; the Hospital of the Protestant Episcopal Church, 80 cases; and Philadelphia Hospital for Contagious Diseases, 7 cases. In the first two institutions named the cases comprised those admitted during the past eight years; in the third, those admitted between 1915 and 1923 inclusive; and in the last, those treated during the past two and a half years.

ETIOLOGY. As in other collected statistics, the etiologic factor in nearly all of our cases was pneumonia, and where the histories failed to show a preëxistent pneumonia the empyema could always be traced to some respiratory disturbance such as pertussis, influenza or some other bronchial affection. Indeed, it is only reasonable to believe, also, that an unrecognized pneumonia had existed in these cases. In one instance in this series the empyema was secondary to traumatism to the chest wall, and in another case it was supposed to have followed a tonsillectomy done twenty-three days previously, although in this latter case there was a strong suspicion of bronchopneumonia. The relationship of empyema to the contagious diseases will be discussed separately.

ONSET. The duration of the illness before applying for hospital aid varied from six days to two months or more. The onset in many cases was obscure, the first symptom being dyspnea sufficiently urgent for the attending physician to suspect an empyema and to send the child to the hospital, where aspiration frequently revealed the presence of pus and confirmed the diagnosis.

The acute cases usually show high fever, cyanosis, rapid pulse, dyspnea and sometimes delirium, which is not at all uncommon in infants. In the subacute cases, or in the small well-circumscribed pneumococcic empyemas, the above symptoms may be entirely absent, and in their place there will be loss of weight and appetite, a hacking cough, occasional diarrhea, etc. If a previous history of pneumonia can be established, this will greatly facilitate the diagnosis.

DIAGNOSIS. The typical case is that in which there is a return of fever after the pneumonia crisis, with dyspnea, rapid pulse, cyanosis and displacement of the heart. Empyema

should be suspected in pneumonia when the temperature falls but does not quite reach the normal and then begins to oscillate above the normal temperature line; also in cases where the percussion note becomes more flat and there is an extension of the dullness anteriorly. The *x*-ray is usually of great assistance in locating the empyema and in determining the condition of the lungs themselves. The final and absolute diagnosis, of course, rests with aspiration and the demonstration of pus. In using an exploring needle it must be remembered that the size of the bore of the needle and the location and consistency of the pus may, for mechanical reasons, yield a dry tap when pus is really present. In one case in the series (an apical empyema), the *x*-ray and aspiration both failed to be of aid in the diagnosis. The child, fortunately, later developed an empyema necessitatis, pointing above the scapula.

In children, pseudocrises are not common, nor is the termination by lysis a frequent occurrence except in debilitated infants. While there may be such a thing as delayed resolution in the pneumonia of childhood, we have never seen it. Our experience has been that these cases always turn out to be empyema.

The empyema in nearly all of the series under discussion was of the massive type, localized toward the base of the thorax, posteriorly. Interlobar and small encapsulated empyema and collections of pus high in the thorax, either posteriorly or anteriorly, were rare in this series.

CAUSE. What causes empyema? According to Moschowitz, it is due to the rupture of a subpleural abscess. Other authorities attribute it to extension of the original disease process by the way of the lymph channels from the hilum of the lung peripherally. We believe that pleurisy complicating a pneumonia from the onset represents a strong causal factor. In our experience the pneumonias with an early pleurisy have shown a greater tendency to pyothorax than the simple pneumonias without that complication. One case in this series was probably metastatic, following a furuncle. The child developed a septicemia, osteomyelitis of the ribs and radius, and an empyema. This was one of the fatal cases in the series.

INCIDENCE. Of the total number of 256 cases of empyema admitted to the surgical services of the Episcopal Hospital during the years studied in this series, 80 (or 31.8 per cent) were children under twelve years of age. Sex, age incidence and mortality of the entire series appear in the following tables:

TABLE I.—EPISCOPAL HOSPITAL

Age.	Male.	Female.	Total cases.	Right side.	Left side.	Died.	Mortality, per cent.
1 year or under .	1	0	1	1	0	1	100.0
2 years or under .	7	5	12	9	3	2	16.6
3 years or under .	10	7	17	10	7	4	23.5
4 years or under .	7	3	10	9	1	1	10.0
5 years or under .	5	1	6	3	3	0	0
6 years or under .	5	4	9	5	4	0	0
7 years or under .	8	0	8	4	4	2	25.0
8 years or under .	2	3	5	4	1	1	20.0
9 years or under .	4	1	5	3	2	1	20.0
10 years or under .	1	1	2	1	1	0	0
11 years or under .	2	1	3	3	0	0	0
12 years or under .	1	1	2	1	1	0	0
Totals . . .	53	27	80	53	27	12	

In this series there were 2 cases of empyema necessitatis. There were 12 deaths, or a 15 per cent fatality. Of the total cases, 66.24 per cent were right-sided, and curiously enough, there was the same percentage of males.

TABLE II.—ST. CHRISTOPHER'S HOSPITAL

Age.	Male.	Female.	Total cases.	Right side.	Left side.	Died.	Mortality, per cent.
1 year or under .	6	3	9	5	4	5	55.5
2 years or under .	23	9	32	19	13	9	28.1
3 years or under .	13	5	18	11	7	4	22.2
4 years or under .	11	6	17	9	8	3	17.6
5 years or under .	2	4	6	4	2	1	16.6
6 years or under .	2	2	4	3	1	1	25.0
7 years or under .	3	0	3	2	1	0	0
8 years or under .	4	1	5	2	3	0	0
9 years or under .	1	1	2	1	1	0	0
10 years or under .	2	0	2	2	0	1	50.0
11 years or under .	0	1	1	1	0	0	0
12 years or under .	0	2	2	0	2	0	0
Totals . . .	67	34	101	59	42	24	

This table shows a mortality of 23.7 per cent, of which 67 patients were males. The greatest number of these cases occurred during the second year of life. Four children were under six months of age, with one recovery.

TABLE III.—MARY J. DREXEL HOME

Age.	Male.	Female.	Total cases.	Right side.	Left side.	Died.	Mortality, per cent.
1 year or under .	6	0	6	1	5	3	50.0
2 years or under .	14	8	22	10	12	5	22.0
3 years or under .	5	6	11	8	3	0	0
4 years or under .	10	0	10	7	3	0	0
5 years or under .	5	5	10	4	6	1	10.0
6 years or under .	12	5	17	6	11	3	17.6
7 years or under .	3	4	7	5	2	1	14.2
8 years or under .	4	1	5	5	0	0	0
9 years or under .	3	0	3	1	2	0	0
10 years or under .	4	2	6	3	3	1	16.6
11 years or under .	0	0	0	0	0	0	0
12 years or under .	6	0	6	3	3	1	16.6
Totals . . .	72	31	103	53	50	15	

In this series there was one case of bilateral empyema, the child recovering.

These tables show a preponderance of cases in males, and a greater number on the right side. The greatest number of cases occurred during the second year, and the same year of life shows relatively the highest death-rate, although it was absolutely highest during the first twelve months of life.

TABLE IV.—PHILADELPHIA HOSPITAL FOR CONTAGIOUS DISEASES

Disease.	Cases.	Broncho-pneumonia.	Lobar pneumonia.	Empyema.	Death from empyema.
Diphtheria .	5,805	83 (1.4%)	7	1 (0.017%)	0
Scarlatina .	5,081	12 (2.0%)	5 (0.1%)	5 (0.1%)	2 (40%)
Measles . .	352	36 (10.2%)	0	0	0
Pertussis . .	271	30 (11.07%)	0	0	0
Variola . .	275	9 (3.2%)	0	0	0
Varicella . .	85	0 (0%)	0	0	0
Scarlatina } .	301	7 (2.3%)	0	1 (0.3%)	1 (100%)
Measles } .					
Diphtheria } .	67	10 (14.4%)	0	0	0
Measles } .					
Pertussis } .	15	3 (20.0%)	0	0	0
Measles } .					
Anthrax . .	38	2 (5.1%)	0	0	0
Totals .	12,290	192	12	7	3 (42.8%)

One patient with bilateral empyema in this series recovered.

The statistics from the Philadelphia Hospital for Contagious Diseases are somewhat different from those of the general hospitals. The cases are those occurring during the past two and a half years among 12,290 total admissions for scarlet fever, diphtheria, measles, pertussis, variola, varicella and certain mixed infections, as tabulated above. Of the total number, 192 were complicated by bronchopneumonia, and 12 by (or intercurrent with) lobar pneumonia. Only 7 of the entire series developed empyema, 6 of them being within the age limits considered in this paper.

It thus is evident that empyema is rather a rare complication of what are known as the communicable diseases but when it does occur, it invariably follows a complicating or an intercurrent lobar pneumonia or a complicating bronchopneumonia, and complicates scarlet fever more frequently than any other disease of this group. This experience is substantiated by other observers. Pfaundler and Schlossman (1924), for example, say that "pneumonia is a comparatively rare complication of scarlet fever, and if this complication sets in at the height of the disease (and here usually only small children are concerned), the prognosis becomes considerably more grave, because it almost invariably leads to infection of the pleura, which in turn is followed by an empyema, very often fatal." This occurred in 50 per cent of our series. The pus in these cases usually was of the thin, streptococcic type. Griffith likewise considers pleurisy with an effusion, often purulent, as only an occasional complication of scarlet fever. With regard to rubella, diphtheria and pertussis, it is generally conceded that serous or purulent pleurisy may occasionally arise to complicate one or the other of these conditions.

TREATMENT. Much depends upon an early diagnosis. This is of the greatest importance, and includes not only the recognition of pus in the chest, but the presence or absence of further pneumonia processes, and the *vis a tergo* of the patient, whether he is in a negative phase from toxemia or has reacted sufficiently well from the toxemia to stand operation. Even in the presence of pus, immediate operation was not always resorted to, for pneumonia was at times still present, or the physical condition was such that less drastic measures had to be used until it was safe to operate.

The question of operation in these cases bears a marked resemblance to the question of immediate operation in acute appendicitis with diffuse peritonitis, where, also, it often is better to wait for localization and a better state of resistance on the part of the patient.

Resolution of the pneumonic process was always awaited before operation. Thin, streptococcic pus was nearly always treated by aspiration, for a resection in the presence of this organism, especially in infants, usually ends fatally, especially if the case is of an acute, fulminating type.

In all our hospital services the *modus operandi* was pretty much the same, varying only in certain minor details. The anesthetic was either ether, gas and oxygen, ether-gas-oxygen or local. We believe local anesthesia or gas-and-oxygen to be the best. Local anesthesia was successfully used upon a baby only four months of age, and to good advantage in older children. Two cases of rib resection under ether anesthesia were followed by pneumonia. The general method pursued was to overcome the respiratory and circulatory embarrassment by repeated aspirations until the effusion became thickened, formal resection being deferred until nature had an opportunity to prevent its spread by the establishment of barriers in the form of adhesions.

Usually the eighth, ninth or tenth rib in the postaxillary line was resected, and fenestrated rubber tubes, usually two in number, were inserted into the wound at the time of operation. It is very important that these tubes be placed so that drainage will be at the most dependent point. If a patient was considered too sick for immediate rib resection, either on account of an existing pneumonia or lack of localization of the empyema or septicemia, aspiration was performed, or a trocar was plunged into the pleural space under local anesthesia, and after threading a catheter through the trocar the latter was removed. The drainage thus afforded was continued until formal rib resection was deemed expedient.

In about one-third of the cases at the Episcopal Hospital, irrigations were done at the time of operation, and in about 9 per cent after operation. We have gained the impression, however, that irrigation neither hastened convalescence nor

shortened the stay in the hospital, nor did it do any material harm, although we have seen cases where irrigation as an after-treatment seemed to cause quite a septic temperature, for when the irrigations were discontinued the temperature speedily came to normal.

The sudden withdrawal of large amounts of fluid from the chest was avoided at all times, in order to prevent sudden changes in circulation and respiration, lest the heart and lungs might be unable to adjust themselves to these changes of volume and pressure. Violent paroxysms of coughing and cardiac irregularities were observed when pus in large quantities was withdrawn too suddenly. The patients were placed upon the abdomen during the resection, because in this position there is less liability to paroxysmal coughing and the lungs are given a much better opportunity to function.

Both in infancy and in childhood pneumococcic empyema with thick pus and fibrin will usually be found to be walled-off and localized by dense adhesions. In this type of case, rib resection is indicated; the empyema cavity can then be gently swabbed out with gauze and all the loose fibrin clots removed. The massive type, however, requires aspiration before resection. The prognosis in these types of pneumococcic empyema is very favorable, the mortality being only about 5 per cent.

Our experience at the Philadelphia Hospital for Contagious Diseases was most instructive. The cases in which a resection was done all ended fatally, whereas those that were aspirated or had intercostal drainage all recovered. The type of operation depended also upon the character and amount of pus obtained by aspiration. Thin, streptococcic pus, such as often follows influenzal pneumonias in infants, was always a contraindication for resection, not only because of the danger of collapse of the lung due to lack of adhesions, but also because of the risk of secondary infection which in some instances sets up a more acute inflammation than the original one.

As soon as feasible the fenestrated rubber tubes can be shortened a few centimeters at a time and finally removed. If there are no signs of returning pyrexia the wounds can be allowed to heal. Measures to expedite drainage are in order,

such as the Woulfe bottles, blowing soap bubbles, toy balloons, whistles, etc. Young children, however, cannot be induced to use these devices.

No patient of the entire series developed acute osteomyelitis or necrosis of the ribs. We have never tried primary closure after emptying an empyema cavity, for we believe the procedure is wrong in principle. Secondary infection of an empyema wound may occasionally occur, but this incidence can be reduced to a minimum by careful attention to asepsis in dressing the wound. As already indicated, we have found irrigation to be of no great value; in fact, our experience with Dakin's and other solutions highly recommended by some authorities has been, to say the least, disappointing.

The treatment of empyema should be directed, not only to relief of the condition and prevention of death, but also to avoidance of prolonged drainage. Close attention, therefore, should be paid to other measures, such as forced feeding, to overcome the tremendous loss of nitrogen due to the toxemia. The diet should be rich in calories, as recommended by Captain Richard Bell, of the Empyema Commission, the value of whose method has been well established by clinical observation. Every care should be taken to prevent scoliosis, which occurs more frequently following empyema than is commonly supposed. Two cases recently have come to our attention in which institutional care at the orthopedic department of St. Christopher's Hospital has been made necessary. In both cases the patients had previously been of normal posture, the deformity having been produced by rib resection for empyema.

SUMMARY. 1. Thirty-one per cent of all empyemas occur in children under twelve years of age. The mortality rate is 17 per cent. Empyema occurs more frequently in males and upon the right side. In children under twelve years, the greatest number of cases occur during the second year of life.

2. Empyema follows some form of pneumonia, except in rare instances, when it is due to trauma. Primary purulent pleurisy was not seen in this series.

3. Empyema is a rare complication of the communicable diseases, having been observed only seven times in 12,290

cases. It occurs most frequently in scarlet-fever cases. However, when present, its mortality rate is high (50 per cent).

4. Operation for empyema should not be done until the pneumonia processes have resolved and until the pus has thickened (not in the presence of thin, streptococcic pus), permitting adhesions to form and the pus to become encapsulated. This at the same time gives the patient a chance to react from the negative phase produced by the toxins of the disease.

5. If rib resection is not immediately advisable, repeated aspirations will serve until the factor of safety is reached and operation may be attempted.

6. If there is any choice of anesthesia, it lies upon the side of local infiltration.

7. Aspiration of about one-third the fluid is advisable before operation, so as to equalize the circulatory and respiratory mechanism. At the time of resection the pus must not be too quickly withdrawn.

8. Irrigation has little or no helpful influence upon the period of convalescence.

9. The empyema cavity should be drained at the most dependent point.

10. In postoperative treatment the most important factors are: sufficient drainage, food rich in calories, measures to promote lung expansion and the prevention of deformities.

11. Recovery from empyema is slow, and the disease cannot be considered cured until the sinus is healed and there are no signs of further trouble attributable to the empyema.

LIPOID NEPHROSIS: TWO ILLUSTRATIVE CASES*

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FOR a concise but inclusive description of the syndrome commonly known as lipoid nephrosis I can find no better terms than Elwyn's definition in his monograph on nephritis: "A disease characterized by an insidious onset, a chronic course, edema, oliguria, albuminuria, changes in the protein and lipoids of the blood and the deposit of lipoids in the kidney; it occurs alone or in combination with diffuse glomerulonephritis or with amyloid degeneration of the kidneys." In the older writings on chronic parenchymatous nephritis are to be found descriptions of numerous cases which conform well to the syndrome as we now know it. Friedrich Müller, in 1905, coined the term "nephrosis" to distinguish those affections of the kidney which are characterized primarily by degenerative changes, especially in the tubules, from those which are primarily inflammatory or due to vascular sclerosis. Munk, studying those cases, found doubly refractive lipoid granules in the urine and in the cells of the kidney tubules, and applied the term "lipoid nephrosis" to the condition. Volhard and Fahr, about the same time, described similar cases under the name of "genuine" or "chronic" nephrosis. Epstein, whose name is often attached to the syndrome, advanced the idea that the condition is a perversion of general metabolism in which the renal features present only one aspect, and he introduced the high-protein diet in treatment. Eppinger, on empiric grounds, used thyroid gland in treatment before studies showed that the basal metabolic rate is often diminished. Addis believes that the condition is one of the manifestations of nephritis and not a pathological entity. In spite of the many discussions in the literature the true nature of the condition has not been explained.

* Read before the Episcopal Hospital Staff Meeting, October 16, 1929.

REPORT OF CASES. CASE I.—A married white man, aged forty-five years, was admitted to the service of the writer on August 6, 1928. In subjectively perfect health until two months before, he had since suffered from nausea, vomiting and weakness; his feet had been swollen since the onset of his symptoms. There were no other complaints except a little vertigo and a tendency to constipation. He had had nocturia once per night for a long time. His urine was said to have boiled solid with albumin on a recent occasion. He had been on a low-protein, low-salt diet since onset; he had had a chronic discharge from one ear since early manhood; in 1926 he had a chancre. He received extensive antiluetic treatment from his family physician, including large doses of mercuric chloride by mouth ($\frac{1}{10}$ grain t. i. d. for several months).

When admitted there were slight puffiness of the eyelids and edema of both lower extremities, extending to the buttocks. The heart was slightly enlarged to the left and the blood-pressure was 155 systolic and 106 diastolic. The retinal vessels showed a moderate increase of the light-reflex, but no marked sclerosis; there were no retinal hemorrhages nor exudates.

For some time after admission he was greatly troubled by nausea and vomiting, so that he was unable to take food. The previously noted edema increased and involved the scrotum. Signs of a right pleural effusion appeared. The urine always contained a "cloud" or a "heavy cloud" of albumin and numbers of hyaline and granular casts; the specific gravity ranged from 1.006 to 1.035. The phenol-sulphonephthalein excretion was 25 per cent in two hours. The blood-urea nitrogen was 24.6 mg. per 100 cc. on admission and it rose to 38.2 mg. per 100 cc. during the time in which nausea and vomiting were most marked and the edema was increasing; just before the patient's discharge from the hospital it had fallen to 16.6 mg. per 100 cc. in spite of a higher protein intake. The blood creatinine ranged between 1.8 and 2.6 mg. per 100 cc. The uric acid was 4.1 and 5 mg. per 100 cc., the cholesterol 340 mg. per 100 cc., and the plasma CO₂ content 65.5 volumes per cent. On three determinations the serum albumin was 1, 1.3 and 1 gm. per cent, and the globulin 2.4, 2.3 and 4.2 gm. per cent. The basal metabolic rate on two successive days was -11 and -12 per cent. The blood-count was not significant except that once there was a leukocytosis of 14,800. The blood Wassermann was negative. When the pleural effusion was aspirated, half a liter of slightly milky fluid was obtained; its specific gravity was 1.003.

The patient was given a diet fairly rich in protein but not definitely measured. No thyroid preparations or diuretics were

used. His nausea subsided, his edema rapidly diminished, the urinary output increased and he felt greatly improved. At the time of discharge his blood-pressure was 155 systolic and 98 diastolic. After some weeks of rest at his home he was able to return to his occupation. When last heard of, over a year after his admission to the hospital, he was proceeding comfortably, with little or no edema, but with persistent albuminuria, cylindruria and hypertension.

CASE II.—A married white woman, aged thirty-six years, was admitted to the service of Dr. Carson on October 24, 1928. Her chief complaints were shortness of breath and swelling of the legs and abdomen. A month previously she "caught a severe cold in the chest;" soon afterward she developed successively swelling of her left leg, dyspnea and swelling of her right leg. A physician examined her urine and told her that she had Bright's disease. Her abdomen began to enlarge and she was very uncomfortable, dyspneic and "cold all over her body." Her lips and finger-nails were blue. Her urinary output diminished to as little as half a pint per day. During the week before admission, fluid began to ooze first from the left flank and then from the right. She had no serious previous illnesses, but had had a "bronchial cough" as long as she could remember and nocturia for several years. She had two children, aged five and two and a half years.

She appeared very ill and was orthopneic, pallid and drowsy. She exhibited a most remarkable degree of edema. The legs were huge and cylindrical; she could scarcely move them because of their weight. The abdomen was enormously swollen, the flanks bulging far out. The skin of the lower abdominal wall showed many tortuous "channels" extending upward toward the thorax, broader at their lower ends, slightly raised, pale, almost translucent. These were at first thought to be dilated lymphatic vessels, but were probably extremely edematous striae of former pregnancies. Fluid oozed freely from the skin of both flanks, saturating dressings quickly and keeping the bed wet. The trunk was edematous as far up as the armpits and the arms slightly so; the face was not puffy. The eyegrounds were normal.

Her urine always contained a "heavy cloud" of albumin and hyaline and granular casts; the specific gravity was from 1.029 to 1.032; the usual twenty-four-hour volume was about 500 cc. The phenolsulphonephthalein excretion was 45 per cent in two hours. The hemoglobin was 60 per cent (Dare); the erythrocytes 3.5 millions; the leukocytes 12,800, and the differential count normal.

The blood-urea nitrogen was 11 mg. per 100 cc.; the cholesterol 360 mg. per 100 cc.; the serum albumin 2.1 gm. per cent; the globulin 2.36 gm. per cent. The blood Wassermann reaction was negative.

Eight Southey tubes were inserted in the patient's legs and thighs; the drainage in about twenty-four hours amounted to 6650 cc. Under theocin the urinary output reached a maximum of 1250 cc. Her diet was arranged to contain 100 gm. of protein, 50 gm. of fat and 50 gm. of carbohydrate per day. The oozing of fluid from the flanks ceased and the edema of the legs diminished to a slight puffiness. In a few days the edema recurred, diarrhea developed (probably from edema of the intestine), the patient grew rapidly weaker and soon died, apparently from exhaustion. Necropsy permission was refused.

These two cases illustrate the syndrome of lipoid nephrosis in somewhat different aspects. Complicating factors are present in the first case; the nitrogen retention, hypertension and impairment of phenolsulphonephthalein elimination point to a coincident or underlying glomerular nephritis. In the second case, an actual nephritis cannot be proven; I hesitate, however, to term it pure lipoid nephrosis because several features of it are not clear.

I wish to make a few comments concerning the cardinal features of lipoid nephrosis. They must be in the nature of a summary because any full exposition would be beyond the limits both of our time and of our actual knowledge.

The generally accepted explanation of the deceptive pallor which causes these patients to appear more anemic than they actually are is that it is due to constriction of surface capillaries. Such constriction would appear to be an effort to minimize the edema by lessening transudation through the capillary walls. There are three obvious possibilities to aid in explaining the production of the edema: (1) alterations in the composition of the blood; (2) some alteration in the permeability of the capillaries; (3) some change in the protoplasmic chemical composition of the tissue cells so that they absorb more fluid. Such changes in the capillaries and in the tissue cells might be due to the unknown toxin or unknown perversion of cellular metabolism which underlies the condition. The edema fluid itself is dissimilar in composition to that found in

acute diffuse nephritis or in cardiac decompensation; the protein content and, according to some investigators, the chloride content also are lower in the edema fluid of nephrosis. In regard to the albuminuria two principal explanations appear, each more or less correlated with a particular conception of the pathogenesis of the condition. In the urine, albumin vastly predominates over globulin; it may be assumed that the hypothetical causative toxin (which attacks most or all of the body cells in varying manners and degrees) injures the cells of the kidney tubules and of the glomerular capillary tufts, so as to make them more permeable to protein, and especially to albumin because its molecules are smaller than those of globulin. On the contrary, others assume a qualitative change of unknown nature in the albumin which makes it unfit to subserve its normal purposes in the body; it is, therefore, eliminated by the kidney as though it were a foreign protein. Such a change would imply a general perversion of protein metabolism; it is with this in mind that Epstein applies the term "diabetes albuminuricus" to the syndrome.

The loss of albumin in the urine, whatever its explanation, accounts for the lowered amount of albumin in the blood. The "normal" amounts of albumin and globulin in the serum are stated differently by different writers; a consensus of opinion is that the albumin should be between 3.5 and 5.5 gm. per cent, and the globulin between 1.5 and 3.5 gm. per cent. In both of our present cases the albumin is greatly diminished, the globulin either normal or slightly increased. In many of the reported cases the globulin is absolutely as well as relatively increased; Linder, Lundsgaard and Van Slyke believe that this is because it is easier for the body to synthesize globulin than albumin. It seems possible that dietary restriction of protein which was practised in our first case, and which is so common if the situation is not completely studied, may contribute to the deficiency of blood protein. The increased cholesterol content of the blood has been believed to be due to an effort by the body to compensate for the decrease in protein; in our cases the cholesterol is well above the normal of 175 to 225 mg. per 100 cc. The hypercholesterolemia and an increase in blood fibrinogen account

for the fact that sedimentation of the erythrocytes is usually speedier than normal.

The lowered basal metabolic rate which has been stressed in some writings is not especially conspicuous here. It is evidently not an essential feature of the syndrome and may be more apparent than real. These patients are usually undernourished, which of itself decreases the metabolism. A degree of edema which would pass almost unnoticed may yet represent a considerable proportion of the body weight and thus constitute a source of error in the calculation.

The etiology, in spite of all that has been written on the subject, remains unsettled. Epstein believes the condition to be a general metabolic disorder. That, however, does not explain the cause; and it may be suspected that the metabolic aspect has been overemphasized because the lowered metabolism is not invariably found and may even be spurious. Munk believed that syphilis is the most important cause. As our first patient had had a fairly recent syphilitic infection, that may have been a factor; in his case, too, the history of large doses of mercury raised the question of kidney damage by the mercury. Zemp, lately a resident physician in this hospital, has reported a case in which carbon monoxide poisoning shortly before the onset of the nephrosis was believed to have been a causative factor. Talley and Glenn, at the Presbyterian Hospital, have recently reported a case in which the patient was in the habit of taking enormous doses of phenolphthalein to relieve chronic constipation, and this was suspected as the cause. The condition seems often to be associated in some way with pregnancy; the so-called "kidney of pregnancy" constitutes one type of nephrosis, which may be the precursor of the lipoid type. When lipoid nephrosis occurs as a complication of chronic glomerular nephritis the former is almost certainly caused in some way by the latter, although we cannot say just what the determining factor is. However, it is safe to say that the commonest cause of lipoid nephrosis is chronic glomerular nephritis. The suspicion naturally arises that our low-protein diets, prescribed with none too great discrimination in cases of nephritis, may aid in inducing the condition. We may have been too strict in our limitation of protein intake.

In treatment the most important measure seems to be a diet rich in proteins and moderately restricted in fats. The ostensible purpose of the high-protein diet is to remedy the deficiency in blood protein; Elwyn believes, however, that any benefit produced is due to the specific dynamic action of the protein in speeding up metabolism. It is noteworthy that even when patients are symptomatically improved by a high-protein diet their serum protein often continues to be below normal. Desiccated thyroid in rather large doses as recommended by Eppinger, seems sometimes to produce decided benefit. Xanthin diuretics often aid materially in reducing edema, as does also restriction of fluid and salt intake. In cases with glomerular nephritis and a tendency to azotemia the balancing of the protein intake is an exacting problem. One must steer between the Scylla of depletion of blood proteins and the Charybdis of nitrogen retention, and in each individual instance one's only chart is the patient's condition.

Lipoid nephrosis runs a chronic course, and most cases, if not all, eventually terminate fatally. The patients may die of the underlying disease—for instance, chronic glomerular nephritis—or of an intercurrent infection. An especial tendency to pneumococcic infections has been described. The cause of death in our second case is obscure. On the other hand, some patients may improve and continue in comparative comfort for many years, but usually with residual symptoms.

Pure lipoid nephrosis, it is rather generally agreed, does not cause "renal insufficiency" in the usual sense of progressive deterioration of the kidney's ability to perform its ordinary work of excretion. But lipoid nephrosis is more often seen combined with other types of renal disease, especially chronic glomerular nephritis, than alone; and in such combination lipoid nephrosis is probably more common than has been supposed. I remember, as no doubt every one of us does, many pallid, edematous patients with hypertension and azotemia, obviously suffering from chronic nephritis; with the traditional superiority of hindsight, it now appears probable that in some of those cases further study might have revealed the presence of a complicating lipoid nephrosis. I wish to stress the type of patient I refer to—"a pallid, edematous

patient with hypertension and azotemia"—and to urge that every such patient should have the benefit of complete study, especially determinations of the blood proteins and cholesterol. Their chance is none too good at best, but they must have whatever opportunity may be offered by treatment aimed to combat a complicating nephrosis should it be found, as well as the underlying nephritis.

REFERENCES

- Addis, T.: *Am. Jour. Med. Sci.*, 1928, clxxvi, 617.
Cutler, J.: *Am. Jour. Med. Sci.*, 1926, clxxi, 882.
Elwyn, H.: *Nephritis*, New York, 1926.
Eppinger: *Zur Pathologie und Therapie des Menschlichen Oedems*, Berlin, 1917.
Epstein, A. A.: *Am. Jour. Med. Sci.*, 1917, cliv, 638; *ibid.*, 1922, clxi, 167; *Med. Clin. North Am.*, 1920, iv, 145; *ibid.*, 1922, v, 1067.
Fahr, G., and Swanson, W. W.: *Arch. Int. Med.*, 1926, xxxviii, 510.
Linder, Lundsgaard, and Van Slyke: *Jour. Exper. Med.*, 1924, xxxix, 921.
Müller, F.: *Verhandlungen der Deutschen pathologischen Gesellschaft*, Jena, 1905.
Munk, F.: *Ztschr. f. klin. Med.*, 1913, lxxviii, 1; *Med. Klin.*, 1916, xii, 1019.
Talley, J. E., and Glenn, H. R.: *Med. Clin. North Am.* (in press).
Volhard and Fahr: *Die Brightsche Nierenkrankheit*, Berlin, 1914.
Zemp, F. E.: *Jour. South Carolina Med. Assn.*, 1928, xxiv 160.

ANESTHESIA IN 1852 AND NOW

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SURGICAL anesthesia had been in use about six years when the Episcopal Hospital was founded in 1852; the hospital, therefore, came into being at the beginning of a most important era in surgery.

It is difficult for us in this day and generation to comprehend what an ordeal an operation was for patient and surgeon before this great discovery. We get a little better understanding if we trace the records forward from the oldest written traditions which relate the attempts to produce artificial sleep to the present day when we have an anesthetic suitable for each type of case.

The desire to relieve pain is as old as the history of man, but its consummation was long delayed. For many centuries there existed an unending difference between desire and attainment which found expression in the words of Hippocrates, "Divinum est opus sedere dolorem." The oldest traditions relating attempts to produce anesthesia came from the Egyptian, Chinese, Greek and Roman physicians. They were acquainted with the narcotic properties of the juices of certain plants and used them in the form of drinks to relieve the pain of patients undergoing surgical operation. Alcoholic beverages were also used for this purpose.

During the middle ages narcotic inhalations were used in an effort to produce general anesthesia. Sponges soaked in the juices of mandrake root, hemlock, henbane and poppy were used, the patient being made to inhale the vapors to induce sleep.

It is interesting to note that in these ancient times attempts were made to produce local anesthesia. There are two such agents mentioned, one was the dried and powdered skin of the

holy animal of Egypt, the crocodile. This powder rubbed on the skin of the patient was supposed to produce anesthesia. The other was the stone of Memphis which, according to Pliny, produced local anesthesia if rubbed on the skin with vinegar. Another method of producing local anesthesia which was brought down from ancient times was the use of compression on nerve trunks. This method was popular for amputation of limbs and was used until comparatively recent times. In the middle of the sixteenth century, Thomas Bartholinus suggested cold as local anesthesia. His suggestions were forgotten and three hundred years passed before cold was again used. Napoleon's friend and chief surgeon, Baron Larrey, states that the wounded in the battle of Eylau requiring amputation had no sensation in their limbs, the temperature being 19° below zero.

So from ancient times until the middle of the nineteenth century surgeons performed their operations with nothing better for anesthesia than had the ancients. Between the years 1844 to 1847 three great discoveries were made, and from this time until the present day strides have been made in the development of general and local anesthesia, and in the methods of giving them. Let us review, then, the past three-quarters of a century and see what these developments were.

In 1844 William Crawford Long, of Georgia, discovered the use of *ether* as an anesthetic, the use of which had been suggested to him during the so-called "ether frolics," when his associates and himself amused themselves by inhaling ether for its peculiar exciting effect. Under its influence he observed that bruises and injuries sustained during these frolics were not felt. He did not make known his discovery, however, and it remained for William Morton to attract the attention of the medical profession to the effects of ether in rendering the patient unconscious of pain. In 1846, in the Massachusetts General Hospital, a patient was successfully operated upon under ether anesthesia and the news quickly spread around the world. It is said that Morton attempted to patent his discovery under the name of "lethon," but the characteristic odor of ether betrayed his secret. The idea of using ether as

an anesthetic was really suggested to him by a chemist, Jackson, with whom he fought a long and bitter struggle for the honor of the discovery. Both died poor and ill from the effects of disappointment.

Morton made use of a flask for administering ether. In 1862, Clover devised a nosepiece by which the ether could be administered continuously through the operation. Following this, various types of inhalers were to be found on the market. In 1876, Clover devised another inhaler, in which the ether was supplied from a reservoir and breathed back and forth from a bag attached to the facepiece, fresh air being admitted from time to time. In 1877, his portable regulating inhaler added greatly to the convenience and safety of ether anesthesia.

In 1893, the open-drop method of administration was described by Prince. The advantage of this method is a minimum of rebreathing.

In 1903, Gwathmey realized that warm ether vapor was less irritating to the respiratory passages than was cold, and urged that the former be used. In 1902, Coburn constructed an apparatus, including an electric heater, which warmed the vapor. In 1916, Shipway introduced his apparatus, in which the ether container was placed in a hot-water bath so that the vapor reached the body at about 60°.

Although the use of *nitrous oxide* for anesthetic purposes was suggested by Sir Humphrey Davy in 1800, it was not seriously taken up until 1844, when Horatio Wells attempted to demonstrate to the profession its value. Unfortunately, not quite enough gas was given to the patient, and as the operation was begun he jumped screaming from the table, with the result that Wells was hissed from the room and treated as an impostor. Wells continued his experiments, with greater success, but died a few days before the Medical Society of Paris passed a resolution giving him the honor of having first discovered and successfully used nitrous oxide as an anesthetic.

Gas was first sold in large rubber bags, and later in iron bottles. Into a quart bottle 15 gallons of gas were compressed, with the result that the tops frequently were blown off. Finally those difficulties were overcome by marketing the gas in liquid form, contained in strong steel tanks. The first method

of administering gas was from an ox bladder, to which a wooden tube was attached as a mouthpiece. During the administration the patient's nose was held, thus forcing mouth-breathing. It is not surprising that failures were frequent with such measures. Later, facepieces of lead or leather covered with rubber were brought into use. In 1867 J. Marion Sims performed several major operations under nitrous oxide anesthesia. Coleman found that prolonged anesthesia could be better maintained when a small quantity of air was administered with the gas. He anticipated the use of nitrous oxide and oxygen anesthesia by adding a valve to the apparatus for regulating the percentage of air. In 1868, Andrews suggested the use of oxygen with nitrous oxide. It was in this year, also, that the use of nitrous oxide as an inductive agent to ether anesthesia was begun. By this means the amount of ether needed was reduced and the stage of excitement greatly lessened.

In 1847, Simpson created a sensation by his article on the use of *chloroform* as an anesthetic. He contended that it was much less irritating to the mucous membranes than ether, and was much easier to administer. For many years chloroform remained the anesthetic of choice in Great Britain, while in this country ether was preferred. It was not long, however, before deaths due to chloroform anesthesia were reported in considerable numbers. It was believed that these ill-effects were due to the action of the drug on the heart, this in turn being due to its use in too concentrated a form. In 1862, Clovers' inhaler, in which the percentage of chloroform as well as the amount of air could be regulated, was brought into use.

The latter part of the nineteenth century and the first part of the twentieth century were devoted to diminishing the dangers of anesthesia and perfecting apparatus for its administration. Pharmacological and physiological aspects of anesthetics were also studied extensively.

Local anesthesia, as we know it today, may be said to have begun with the discovery of the anesthetic effect of cocaine. In 1884, Koller was able to perform operations on the eye after dropping in cocaine. Hall and Halsted pointed out that the

injection of the nerve trunk with cocaine in any part of its course was followed by sensory paralysis in the entire peripheral distribution of the nerve. This discovery was the forerunner of the neuroregional method of Crile and Cushing and Matas. To Reclus and Schleich belongs the credit of proving the importance of employing more dilute solutions of cocaine, thus considerably reducing its toxicity. In 1904, after much experimenting, Einhorn derived from cocaine a substance much less toxic, called novocaine. Braun made use of novocaine as an anesthetic in the following year.

In 1892, the *infiltration method* of local anesthesia was introduced by Schleich. In this type of anesthesia the sensory-nerve endings of the field of operation were acted upon.

In 1888 and 1889, *spinal anesthesia* was brought into use, having been suggested and first used by Corning. Cocaine was the first drug used for spinal anesthesia. It was soon replaced, however by the less toxic drugs stovaine, novocaine and tropocaine.

Sacral anesthesia was first suggested by Cathelin, in 1900, but not seriously taken up until 1910, when Laewen did much to arouse interest in this method of anesthesia.

Kappis, in 1913, described the use of *splanchnic anesthesia* in operations of the upper abdomen. His theory was to block the sympathetic nerves by injecting the semilunar ganglion. This method has never gained great popularity, owing to the difficulty of administration.

Auer and Meltzer, in 1912, described the *synergistic action* of magnesium sulphate and ether, the former being given intramuscularly, the latter by inhalation. It was found that a small amount of ether, not sufficient in itself to produce anesthesia, combined with magnesium sulphate intramuscularly, would produce a good and prolonged anesthesia.

In 1920, Yandall Henderson drew attention to the fact that carbon dioxide aided in the induction of ether anesthesia, increasing the rate of absorption, requiring a lower concentration of ether, reducing struggling and excitement and offering greater relaxation under light anesthesia. Lundy enumerates several advantages of its use, among which may be mentioned the hastening of elimination of ether, the possibility of inducing

deeper narcosis, and the control of respiratory depression from an overdose of ether.

Rectal anesthesia was first suggested by Roux in 1847 and brought into practice by Pirogoff in the same year. At first, liquid ether mixed with water was used. Cases of melena and death from its use were reported and the method was temporarily abandoned. In 1910, Cunningham, Lacey and Gwathmey tried olive oil as the vehicle instead of water. This method was found to be safe and has gained greatly in popularity, especially in operations where the air passages are involved, in operations upon the neck and head and in obstetrics.

Ethylene was introduced as an anesthetic by Luckhardt and Carter in 1923, and in many clinics throughout the country it is extensively used. It is said by those who use it to have great advantages over other methods of anesthesia in that it is rapidly induced, causes no disagreeable after-effects, is non-irritating to the respiratory mucosa and does not cause labored breathing or excessive secretion of mucus. Pulse and blood-pressure are influenced less by ethylene than by other anesthetics. The recovery period is brief. Whatever its advantages may be, it must be said, however, that the gas is highly explosive and its use carries with it no little element of risk to the patient, operator and attendants. Not a few serious accidents have been reported due to explosions, the gas being ignited by static sparks. The proper grounding of the gas machine is said to have reduced this danger considerably. Gwathmey states, however, that in spite of its disadvantages, there is no anesthetic that can replace it. Nevertheless very few hospitals have been willing to incur the risks incurred by its use.

Recently (1925) Lundy has suggested the use of what he terms a *balanced anesthesia*. He states that small doses of various anesthetics have very rarely any disagreeable or dangerous effects and that small doses of several different anesthetics may together produce anesthesia. For example, he suggests the use of local anesthesia supplemented by nitrous oxide or ethylene, and finally ether is added to produce the desired relaxation.

It will be seen from the above résumé, that in 1852, when this hospital was founded, its attending surgeons had at their disposal three anesthetics—ether, nitrous oxide and chloroform. So far as can be determined ether was the anesthetic commonly used by them. The methods of administration were cumbersome and inaccurate and little or nothing was known of their pharmacological or physiological actions. Though there are no records of the anesthetics given in these early days, it is handed down by word of mouth that fatalities were few.

The contrast, then, is apparent between the anesthetics and the methods of administration in those early days and the present day, when we have a suitable anesthetic for each type of case, an accurate knowledge of its action and every facility for its administration.

During the calendar year of 1928, there were 3636 anesthetics given. In 2574 of these ether was used, in 559 nitrous oxide and oxygen, in 23 nitrous oxide oxygen and ether, in 2 chloroform, in 430 local anesthesia, in 46 spinal anesthesia, and in 2 sacral anesthesia.

Unfortunately there are no records which enables one to compare these figures with those of the early years of the hospital.

SPONTANEOUS LATERAL VENTRAL HERNIA*

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IN presenting this study it is not my intention to claim any originality. The subject of hernia in all its forms has been reviewed repeatedly. Hernia in its many varieties forms a large part of early and modern surgical literature. Gradually the classification into types offered particular subjects for the investigators. Little remains now to be added. Lateral ventral hernia, spontaneous or traumatic, has been written about frequently and fully. Yet the occurrence of a spontaneous ventral hernia offers interest, for because of its rarity it is frequently not diagnosed before strangulation occurs, or until exploratory laparotomy reveals it to be the incipient factor in the production of a series of aggravating symptoms.

Accordingly then it shall be my purpose to report one more case of such a hernia, to review briefly the subject and to point out a few diagnostic difficulties which have been little considered and less admitted.

Definition. The definition of LaChausse is that "ventral hernia is any hernia except a femoral, inguinal or umbilical." He included parainguinal, medial inguinal and supravesical hernia in his classification, and added, "No certain locus can be assigned to them." Mollière wrote that "nearly always these herniæ occur in the linea alba, or a little outside this cicatrix, or in the semilunar line of Spiegel, and on a level with a line from the anterior superior spine of the ilium to the umbilicus." Obviously any hernia of the anterior abdominal wall would be a true ventral hernia. Excluding umbilical, lumbar, and post-operative or traumatic types, we may define spontaneous ventral hernia as one which appears at an abnormal opening in

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the abdominal wall, apparently without explicable reason, but usually presenting in or near the linea alba, or the semilunar line of Spiegel. Our present interest, however, being in lateral ventral hernia, this discussion will be limited to that type of hernia which occurs near the lateral margins of the recti muscles, in that aponeurotic structure commonly known as the semilunar line of Spiegel. More strictly speaking, however, this is not a mere line, for repeated dissections have proved that the blending of muscle layers and ensuing aponeurotic sheaths may not be accomplished through a sudden sharp line of demarcation, and that consequently the so-called Spiegel line may be a fairly broad structure extending a varying distance from the lateral rectus margin. It is, then, in this space that such hernias as we shall discuss occur.

REPORT OF CASE.—K. H., widow, aged forty-five years, was admitted to the Episcopal Hospital, August 25, 1921, in Dr. Ashhurst's service. Family history was negative. She had had the ordinary diseases of childhood and typhoid fever at twenty-four years of age. She had had four children. The menstrual history was negative. Her general health had been always good.

Chief Complaint. Lump in right side. The patient noticed a small lump in lower right side two years ago soon after going to work in a mill where she had to lift heavy loads, sometimes 70 pounds, two or three times a day. The first time she noticed the lump she felt a "click." A month or so later she noticed a lump about the size of a small marble. This grew larger gradually, and became slightly painful. Several times, at various intervals, she noticed that the lump appeared, and she found that upon lying down it disappeared especially when a hot bag was applied to the region. Often the lump was sore to touch and she would be compelled to lie with her leg drawn up. Pain would be cramp-like, but seldom occasioned nausea or vomiting. Usually the lump would be reduced gradually, or as she put it, "by degrees." The site of this swelling was pointed out as being a little above and external to the inguinal region. It was a little larger on admission than when first noticed. It became painful when she exerted herself or sometimes when she would stand up. She had never had much difficulty in replacing it. Sometimes an interval of two weeks occurred without her noticing it. She had suffered constipation, indigestion, belching of gas and occasional vomiting spells for an indefinite time. Other systems were irrelevant.

Physical Examination. The patient is a well-developed, well-nourished, but not obese, white adult female, forty-five years of age. The head, neck, chest and heart are negative. The abdomen is not pendulous. There are striæ over the lower wall (from previous pregnancies). There are no scars, no areas of tenderness or rigidity. The patient points out the site of the lump just above and to the outer side of the right inguinal canal. Repeated examination by manipulation, palpation, and observation failed to disclose any abnormality, the "lump" of which she complained remaining undiscovered as well as any depression or orifice in this region. The genitalia and extremities were negative. Basing the diagnosis strictly upon the patient's history, a diagnosis of hernia was made, though no classification was mentioned.

Operation. August 30, 1921. (Dr. Ashhurst and Dr. Holloway.) Incision 15 cm. long parallel to the fibers of the external oblique above and below the right anterior superior spine of the ilium, exposing the external oblique. The aponeurosis of the external oblique was opened, and beneath it a hernial sac was found the size of a hen's egg, covered with preperitoneal fat. The sac was dissected free, from an opening in the internal oblique and transversalis muscles; which opening was opposite the anterior superior spine, and at least 8 cm. above and to the outer side of the internal ring. Sac opened at its fundus, and excised around its origin from parietal peritoneum. Appendix removed. Meso-appendix sutured for bleeding, stump ligated and buried. Wound then closed in layers with continuous chromic for the peritoneum, interrupted chromic for internal oblique and transversalis as one layer, and continuous chromic for the external oblique and for the skin.

The patient made an uneventful recovery and was discharged sixteen days later. Six weeks after operation the patient reported to me for examination. She was in excellent health and spirits. Her symptoms of indigestion, belching and vomiting had disappeared. She no longer had any attacks of pain or abdominal discomfort. Her general mental attitude was greatly improved.

Historical. History has it that Hippocrates, Avicenna and Galen were familiar with ventral hernia, but the early observers seem to have known only those traumatic in origin. LeDran (1742) gave the first clear discussion of the subject in his *Traité des Operations de Chirurgie*. LaChausse, who gave the first accurate contribution upon the subject of ventral hernia

(1746), states that Celsus devoted an entire chapter to the subject but confused it with umbilical hernia. It remained for Dionis, Garangeot and Heister (1738) to clearly differentiate them. LaChausse distinguished three types of ventral herniæ: (1) Those in the linea alba above and below the navel; (2) those in the lateral epigastric regions; (3) those in the lateral hypogastric regions due to separations of the fibers of the oblique or transversalis muscles. In his studies of the last type, however, he considered those herniæ of traumatic origin only. In 1746 Klinklosch pointed out "Spiegel line herniæ," but in his study he included all types of abdominal hernia. In 1804 Cooper added much to the subject.

Little advance in the study of this particular type of hernia was made then until the latter part of the nineteenth century, and interest in cases assumed only the proportion of curiosity. In 1877 D. Mollière presented a case consecutive with phlegmon and this is accordingly ranked as traumatic in origin. In 1878 Terrier produced his important work upon ventral hernia and in 1879 Mackrocki assembled 86 cases of lateral abdominal herniæ, pointing out favorite sites as vascular exits added by fat. In 1881 Ferrand added his own case to his thesis.

The most elaborate studies seem to have been made within a comparatively recent time. In 1907 Thévenot and Gabourd brought the subject up to date. In 1910 Steimker, and in 1911 Baudoin produced excellent articles. Among many other writers who have studied the subject may be mentioned Gosselin, Belfinger, Graser and Duplay. The latest contribution, in which a most excellent review of the subject by Augé and Simon is achieved, appears in a recent issue of *Revue de Chirurgie* (1921, lix, 297). It is rather striking that apparently little consideration has been devoted to this particular type of hernia by American writers. Perhaps the excellent work by French, German and English writers has made it unnecessary.

Etiology. Berger states that from a standpoint of production two sorts exist: (1) Under a cicatrix or a complete or incomplete rupture of the abdominal muscles; (2) spontaneous.

The cicatricial type is that type which follows trauma to the abdominal wall which may or may not have broken the skin. This type being self-evident need not be further discussed here except to mention that in certain cases in which ventral hernia

may follow trauma after several years have lapsed, the trauma may have been entirely secondary and incidental to the production of the hernia. Belfinger states that traumatic hernia must be completely developed immediately or within a few days after receipt of the injury and that there must have been no predisposition to hernia, no matter of what nature, thus no latent hernia, no empty hernial sac.

These herniæ are most frequently seen in women. Of 29 cases of ventral hernia reviewed by Berger at the Central Bureau (France) there were 23 in women; 20 of these were spontaneous. Berger believes them most common in fat women about thirty years of age. Augé and Simon list 27 women in a report of 47 cases, the youngest being twenty-five and the oldest seventy-two. The youngest male reported was aged one-half year, the oldest forty. These ages are apparently unimportant, however, for they relate only the age of the patient when seen by the surgeon. The date of origin would be more valuable. Unfortunately this is in most cases uncertain. Many of the patients, however, evidently had their herniæ a number of years before presenting themselves. The longest duration we have ascertained is sixteen years. It is evident that the origin may precede the descent of contents into the sac by an indefinite time.

Pathological Anatomy. The site of these herniæ depends primarily upon a solution of continuity of tissues in the abdominal wall, and they are usually limited by the fibrous contour. Peritoneum stretching over a point of weakness finds its way outward, forming a sac. Intraabdominal pressure may be a deciding factor. Rupture of the "posterior fibers and sheath" is essential, says Berger.

Cooper showed that spontaneous ventral hernia usually presents along the line of Spiegel, and particularly at the "junction of the aponeurosis of the transversalis and posterior fibers of the sheath of the muscle." He was the first to point out (1804) that "bloodvessel openings through the abdominal wall" were frequent sites of ventral hernia. Mackrocki, Regnier and Brennan have expressed belief that at this level numerous "hiatus vasculonerveux" (which give perforating "anterior and external" vascular branches and nerves to the abdominal wall) are causative factors.

Ferrand, basing his opinion upon two dissections, states that below the umbilicus there are diastases between the aponeurotic fibers of the transversalis due doubtless to the projections of branches of the deep epigastric arteries which wind between the thin transversalis fascia and the posterior layer of the aponeurosis of the internal oblique. Cooper pointed out also that next these vessels the muscle may be lacking, predisposing to hernia, which, however, may pierce the transversalis at one point and penetrate the overlying area at some distant point. Graser called ventral herniæ outside Spiegel's line "seitlichen Bauchbruch" and believed them due to muscle defects or acquired muscular paralysis. Platner held that herniæ could pierce muscular parts by "separating muscular fibers," but Berger says it would be difficult to find an instance where a true spontaneous ventral hernia had a sac in which the neck was bounded by muscle tissue. B. Schmidt described a preformed peritoneal sac due to the drag of preperitoneal fat as a predisposing cause.

LeDran pointed out repeated pregnancies as the most frequent cause. Augé and Simon have concluded that increased intraabdominal pressure from any cause is primary and they quote Boyer (1822) as saying "of all causes most capable of pushing against the closed abdominal wall, the simultaneous contraction of the diaphragm and the abdominal muscles is most effectual." In vomiting, in labor, carrying burdens, expelling retained urine and accumulated feces, we most commonly find such conditions. Crushes, blows and falls on the abdomen are evidently to be considered. In such cases the trauma sustained may obscure the spontaneous origin of a preëxisting hernia or a sac.

Spiegel line herniæ are usually single. They may be multiple. Berger states that in one patient he saw two on the right side and one on the left, not symmetrically placed.

Diagnosis. Many opinions are expressed as to the ease or difficulty of diagnosis. The patient may have known the presence of an inconstant tumor mass in his side. These cases usually should present no difficulty, especially if the patient is thin, but often the diagnosis is merely presumptive. If the hernia can be seen or felt there is no difficulty. The interstitial type presents more difficulties, and authorities claim that

these may defy detection. In our case no amount of effort or manipulation could cause a hernial protrusion. No depression could be located. Others have found the same difficulty. In the case of a patient who presents himself offering a history of having repeatedly seen a reducible swelling in a particular area, painful, causing digestive upsets, perhaps also vomiting and constipation, the diagnosis of hernia is simple. Many patients have been operated upon without having the hernia discovered at all. In a fat person the hernia may be unreduced but effectually hidden in subcutaneous fatty tissue. A. Mouchet and R. Gouverneur present their case as an illustration of diagnostic difficulty. A soldier, aged twenty-four years, had received a kick in the abdomen just above the groin seven years previously. Sometime later a tumor was noticed by him about the size of a small nut in the region of the injury. It was painless and little noticeable, never causing trouble. An army physician examining him failed to notice it at all. Later, following a fall in a trench the hernia became larger and painful. Operation showed the hernia coming through all three muscles above and to the outer side of the internal ring and containing omentum. These writers believe that the hernia was not due to the kick but that it preëxisted as a congenital diverticulum of peritoneum; that the kick and fall merely contributed to its enlargement and strangulation.

S. Steimker found in the cadaver of a man, aged fifty years, with bilateral inguinal hernia and a supravesical hernia, also a defect in the abdominal wall 6 cm. horizontally and medially from the left anterior superior spine of the ilium; this was a round 3 cm. wide opening with a 7 cm. deep sac protruding. This sac was embedded in the muscles of the abdominal wall. The medial boundary of the sac was the lateral margin of the left rectus. The lateral boundary was not clearly visible or palpable.

Steimker relates that in an unreported case of Braun in a male, aged thirty-eight years, with symptoms of intestinal obstruction, coming to operation, a mass was discovered in the left lower quadrant of the abdominal wall. There was found a strangulated hernia in a sac of peritoneum with an opening the size of a mark piece (German coin) resembling the inguinal ring. (*Beit. z. klin. Chir.*, 1912, lxxxii, 633).

TABLE I.

Number. Sex. Age. Reference.	Size and Site.	Ring.	Contents.	Strangulation diagnosed.	Possible etiology.	Remarks.
No. 1. M., 24. Mouchet and Gouverneur, 1916	Pigeon egg parain- guinal in external oblique and trans- versalis	Buttonhole	Omentum	Subacute, before operation	Kick in groin from horse 7 yrs. previously	
No. 2. M., 20. Williamson, G. H., 1915	"Not large" above and to inner side internal ring	Tight fibrous band	Ten inches ileum	Subacute. Ingui- nal hernia	Duration about 2 yrs.
No. 3. F., Jordan, F., 1883	Tender spot well out- side inguinal canal between umbilicus and groin	Flat sac with 8 inches of bowel	48 hrs. before operation	Previous health good. Diagnosed upon signs of ob- struction. Gave signs of acute ap- pendicitis and ure- teral calculus. Re- covered.
No. 4. M., 69. Teale, T. P., 1842	Left abdominal wall between umbilicus and iliac spine	Tendinous on mesial side. Soft on lateral side	Colon and ad- herent omen- tum	Before operation	Died 12 hrs. after operation. Colon intensely inflamed
No. 5. F., 25. Gosselin, 1881	Thinned out space between umbilicus and anterior supe- rior spine	Several attacks of subacute. Before opera- tion	Coughing	Noticed only few days.
No. 6. M., 63. Terrier, 1878	Left border rectus above internal ring. Sac would admit tip of finger	Annular tightly constricted	2-3 cm. gut	After operation	Six days of pain, occasional vomit- ing. No bowel movement, no fla- tus.
No. 7. M., 48. Robinson, B., 1914	Left side above in- guinal canal	Little finger	Knuckle small gut	Before operation	Noticed 2 yrs. espe- cially in winter when had bron- chitis.

No.	8. M., 53. Coley, Wm., 1909	McBurney's point	$\frac{7}{8}$ inch in diameter, firm	Loop cecum	Before operation	Horse fell on him 7 yrs. previously	Indefinite symptoms of pain in region of appendix shortly after accident, lasting about 4 years.
No. 9. F., 66. Baudoin, 1911	38 by 34 cm. in 3 pockets	Level of umbilicus in left lateral abdominal wall all tight	Colon, cecum appendix. Small gut, omentum. Partly adherent	In parts. Upon sight	Lifting weights	Duration 6 yrs. Umbilicus orifice normal when examined. Recovered.	
No. 10. F., 44. Moller, H., 1919	Internal to iliac spine 4 cm. in diameter midway to rectus border	Adherent omentum	Reducible tumor 15 yrs.	
No. 11. M., 72. Moller, H., 1919	Sac 7.8 cm. long to mesial side muscle fibers of left internal oblique and transversalis	Adherent omentum	Before operation	Operated 1908 for double inguinal hernia.	
No. 12. F., 57. Macewen, John A. C., 1907	Sac 1½ inches long but very distensible midway <i>vs.</i> umbilicus and symphysis in right semilunar line	Narrow	Empty when operated	Complained of pain 2 yrs. previously to operation complicated with small umbilical hernia.	
No. 13. F., 80. Robinson, H. B., 1907	Right iliac fossa, above inguinal canal	Adherent omentum	Signs of obstruction before operation	Symptoms 4 days. Found external to deep epigastric artery.	
No. 14. M., 25. Barthélemy, 1919	In right iliac region just above Douglas' fold	Before operation	Appeared suddenly while lifting heavy joist	Existed 2 to 3 mos. before operation. Easily reducible.	
No. 15. F., 47. Watson, 1919	Between gall-bladder and appendix	Adherent omentum	Repeated attacks subacute	Duration about 5 yrs.	
No. 16. M., 35. Brennan, 1899	Right semilunar line	Intestine and omentum	Appeared suddenly while at work.		
No. 17. F., 34. Holloway, 1921	8 cm. above and to outer side of right inguinal ring	Empty	No. At operation	Noticed first while lifting moderate weight	Duration about 2 yrs.	

It is apparent then that these herniæ may exist for an indefinite time unobserved and absolutely without symptoms. The patient may recognize a disappearing painful tumor, but he usually does not understand its significance. He comes to the surgeon because he associates the tumor with a certain amount of pain, burning or tearing in his abdomen which hinders his work. Usually the tumor will disappear upon lying down.

Palpation may reveal merely a painful spot. Sometimes a tumor mass may be felt which upon pressure reduces with an audible gurgle; or the finger may locate an orifice.

If the patient leans forward the tumor may appear. Anything to increase intraabdominal pressure may cause the hernia to protrude. Often, however, all efforts fail and a presumptive diagnosis is made only upon subjective symptoms.

These herniæ are especially liable to incarceration or strangulation. The extremely distensible sac with a very narrow neck and orifice favors such a result. Gangrene then supervenes rapidly. Most of the cases reported have been found to have been completely or partially strangulated at one time or other, sometimes several times. But even as such, they are sometimes not clearly defined. Cases are reported where acute appendicitis, pyelitis, and cholecystitis have been confusedly diagnosed. Inguinal hernia may be the preoperative diagnosis. No diagnosis at all has preceded some operations where the symptoms of obstruction have led the surgeon to believe that exploratory laparotomy was necessary and justifiably so.

Treatment. Operation offers the only hope of permanent cure. It is to be substituted by conservative treatment only in cases in which any operative procedure is contraindicated. Operation consists in freely exposing the sac by incision, complete excision of the sac and obliteration of the orifice. The overlying structures are then to be closed in layers without necessarily overlapping the fascial or muscle plates. Symptomatic relief may be secured by wearing a tight abdominal belt. In children with diastasis of the recti adhesive straps may be effectual as in umbilical hernia. Usually such treatment is merely palliative.

In cases where the location of the hernia is indefinite we may accept the patient's idea of the location as a key to the situation and make our incision accordingly; though some operators prefer to make a median laparotomy incision and search out the hernia from within, especially when the hernia is concealed by a pendulous or obese abdomen, or thick muscle walls.

In the accompanying table are recorded 17 cases of lateral ventral hernia (including that reported herewith), 11 of which are not included among those tabulated by Augé and Simon in their recent paper. (*N. B.*—Cases Nos. 1, 2, 7, 9, 10, 11, 12, 14, 15, 16, 17.)

REFERENCES

- Augé and Simon: *Rev. de chir.*, Paris, 1921, lix, 297-316.
 Baudoin: *Thèse de Paris*, 1911-1912, vol 2, p. 269.
 Barthélemy: *Bull. et mém. Soc. de chir. de Paris*, 1919, xlv, 1313-1319.
 Brennan: *La revue méd.*, Montreal, 1899-1900, iii, p. 337.
 Berger: *Trait. de chir.*, Paris (Duplay and Reclus, editors), 2d ed., 1918, vol. vi, p. 343.
 Borchgrevink: *Die Hernien und ihre Behandlung*, Jena, 1901, lxiv, 159-192.
 Colcord: *Pennsylvania Med. Jour.*, 1918, xxi, 672.
 Coley: *Annals of Surgery*, Philadelphia, 1909, ii, 246-249.
 Cooper: *On Hernia*, London, 1804.
 LeDentu and Delbet: *Hernies*, Paris, 1908, p. 352.
 Gosselin: *Gaz. méd. de Paris*, 1881, p. 123.
 Gosselin: *Hernies abdominales*, Paris, 1865, p. 323.
 Gibney and Walker: *Twentieth Century Pract. of Med.*, New York, 1897, vol. ix.
 Graser: *Handb. d. prakt. Chir.* (v. Bruns, Garré. u. Kuttner), Stuttgart, 1913, Bd. v, S. 572.
 Jordan: *Birmingham Med. Rev.*, 1883, xiii, 72-73.
 LaChausse: *Dissert. chirurg. de Hernia ventral.*, 1746. *Halleri disput. chir. selecta*, tom. iii.
 LeDran: *Traité des operations de chirurgie*, Paris, Osmont, 1742.
 McCready: *Treatise on Ruptures*, Philadelphia, 1893, p. 258.
 Moller: *Hospitalstidende*, 1919, lxii, 553.
 Macewen: *Lancet*, London, 1907, ii, 1081.
 Mouchet and Gouverneur: *Paris méd.*, 1916, vi, 351. (Same abstracted in *New York Med. Jour.*, 1916, i, 1047.)
 Mollière D.: *Bull. et mém. Soc. de chir., de Paris*, 1877, iii, 278-284.
 Robinson: *Brit. Jour. Surg.*, London, 1914, ii, 336.
 Robinson: *Brit. Med. Jour.*, 1907, ii, 781.
 Stühmer: *Beitr. z. klin. Chir.*, 1910, lxvi, 113-135.
 Steimker: *Beitr. z. klin. Chir.*, 1912, lxxxii, 633.
 Schmidt: *Hand. d. allgem. u. spez. chir.* (v. Pitha-Billroth), 1882, Bd. iii, Abt. 2 A.
 Sultan: *Atlas u. Grundniss der Unterleibsbrüche*, München, 1901.
 Teale: *On Abdominal Hernia*, London, 1846, p. 356.
 Terrier: *Bull. et mém. Soc. de chir. de Paris*, 1878, iv, 361.
 Thévenot et Gabourd: *Rev. de chir.*, Paris, 1907, xxxv, 568-585.
 Watson: *Memphis Med. Monthly*, 1919, xl, 461.
 Williamson: *Surg. Jour.*, Chicago, 1915-1916, xxii, 197.

DISEASES OF THE GASTROINTESTINAL TRACT THEN AND NOW

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IN the year 1852, Louis Pasteur was a young man of thirty years, engaged in his researches upon crystallography. To the medical profession of that time bacteria and infections were unknown and little was known of the chemical and physiological aspects of disease. Needless to say, gastrointestinal diseases were diagnosed and treated without the *x*-ray, stomach tube or the various chemical and microscopic examinations which are today considered a necessary part of the study of such cases. The names of the gastrointestinal diseases treated in the early fifties throw an interesting light upon the state of medical progress of the time. In 1853, out of a total of 180 admissions to the hospital, of which 102 were medical, 14 cases were regarded as gastrointestinal in nature. Of these 5 were classified as "dysentery" (naturally neither the term amebic nor bacillary were known), 5 as "dyspepsia," 2 were cases of "diarrhea" (which must have been difficult to differentiate from "dysentery") and 2 were "diseases of the liver." During the next year there are found the following additional diagnoses: peritonitis, cancer of the stomach, cramps, worms, enlargement of the spleen, jaundice, colica pictonum and cholera. While the meager records of the day do not enable one to say whether the "cholera" was of the Asiatic variety or merely "cholera morbus," which we should now designate as "acute gastroenteritis," yet it probably was the former, for the patient died. Furthermore, it is known that an epidemic of cholera, starting in India in 1846, reached this country in 1848, and gradually spread over the American continent during the ensuing years. The cause of cholera was unknown until 1883, when it was discovered by

Koch to be a spirillum. It is interesting to note, however, that a few of the prophetic minds of the day considered cholera to be a contagion, one writer of the period¹ suggesting the possibility of a minute organism being the cause of the disease, and referring to carriers of the disease who are themselves healthy.

Relatively more patients entered the hospital for digestive disturbances in the early days than in recent years. For example, in the years 1853 to 1856 inclusive, 8.8 per cent (94 out of 1070) admissions were for gastrointestinal disturbances as against 2.5 per cent (113 out of 4534) admissions in 1915.

Chronic gastritis, gastric ulcer and cancer were recognized, but often confused. It was taught that prolonged gastritis resulted in cancer or ulcer of the stomach, and it was believed important, therefore, to differentiate between dyspepsia and gastritis, this differentiation being based partly upon symptoms and partly upon the appearance of the patient, his tongue and pulse. In a textbook of the day² we find that the stomach was known to secrete hydrochloric acid and it was believed by some that the pain of ulcer was dependent upon the acidity. (This theory of the causation of pain in ulcer is held by many today.) It was, furthermore, known from post-mortem findings that spontaneous healing of ulcers could occur. Pain related to the taking of food was recognized as of diagnostic significance by at least one clinician of the day.³

Thirty-one years before this time, Napoleon Bonaparte had died of cancer of the stomach.⁴ Although this keen observer and man of diverse genius had diagnosed his own malady before his death,⁵ yet it must be admitted that the diagnosis of cancer of the stomach was not ordinarily made by the physicians of the early days until cachexia and a palpable mass made their appearance. Perhaps it was just as well that such should be the case, since neither surgery nor x-ray were available to benefit the patient after the diagnosis had been made.

Ulcer statistics of the early days are, of course, worthless, since the diagnosis could only be made at the autopsy table, and, even then, ulcer of the stomach was doubtless often confused with cancer. Nowadays, utilizing all the means at

our disposal, especially the x-rays, it is usually possible to arrive at a correct diagnosis of ulcer before autopsy and before operation.

Having made the diagnosis of peptic ulcer, we find ourselves torn between conflicting ideas as to treatment. In certain types of cases, it is true, agreement exists: for example, it is agreed by all that in cases of perforation or obstruction immediate operation is necessary; on the other hand, treatment in those cases of ulcer whose only clinical manifestation consists in periodic attacks of pain is still a matter for contention, many surgeons maintaining that such cases should be operated upon as soon as the diagnosis is made, while practitioners of medicine commonly hold that medical treatment should be given a more or less prolonged trial. The immediate mortality in 58 cases of uncomplicated peptic ulcer operated upon in the years 1924 to 1927 inclusive was 5.2 per cent (3 deaths). One case with hemorrhage died, which if included would give a mortality of 6.8 per cent. These figures are very similar to those reported from other clinics.

At the present time neither the cause nor the cure for peptic ulcer is certainly known. I venture to make the prediction, however, that when the cure shall be discovered it will not be surgical but medical.

BIBLIOGRAPHY

1. Watson, T.: Practice of Physic, Philadelphia, 1850, pp. 807 and 808.
2. Watson, T.: Practice of Physic, Philadelphia, 1850, p. 778.
3. Flint, A.: Practice of Medicine, Philadelphia, 1866, p. 341.
4. Antommarchi, F.: Mémoires du Docteur Antommarchi sur les Derniers Momens de Napoléon, Bruxelles, 1825, p. 244.
5. Ludwig, E.: Napoleon, New York, 1926, p. 266.

ABDOMINAL INCISIONS—THEIR MAKING AND CLOSURE*

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MOYNIHAN says: "I do not think, though much has been written, it is adequately recognized that the steps in the making and in the repair of the abdominal wound are of the very greatest importance. I doubt if it is an exaggeration to say that the circumstances connected with the incision are among the most important in the whole range of abdominal surgery. For, if the incision be improperly made, by free division of muscle fibers and the wilful and unnecessary severing of nerve trunks, a weakened area is left in the belly wall, the result of which may be of even greater severity than the condition which first made operation advisable. Too great care therefore cannot be exercised in the proper choice of a method of incision and of the means of its securest closure. It is a cardinal rule that there shall be no division of muscle fibers unless it is absolutely necessary for a sufficient exposure of the operation field; muscle fibers are to be displaced or separated without injury to nerve supply, never to be cut."

I have for some years past been interested in the defects of the abdominal wall, the result of operative incisions, and after observing a goodly number of cases from other clinics I am of the opinion that there is a general tendency to disregard this step in abdominal surgery. By observing a few simple rules it is possible to leave the abdominal wall as strong and free of defects as it was before operation. There are instances, more especially in cases of malignancy, when the operator is justified in sacrificing the structure of the abdominal wall in order to

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give sufficient exposure. But it is the usual case to which I refer and not these exceptions.

It does not seem necessary to go into the detailed anatomy of the abdominal wall for we are all familiar with it, but let us consider briefly the structures which concern us most in the making of an incision. Namely, the muscles, the bloodvessels and the nerves.

The muscles of the abdomen, the three oblique and the rectus on either side have various functions: support of the abdominal viscera, accessory to respiration, aiding in defecation, micturition, and parturition, flexion and rotation of the pelvis and trunk. It is evident then that injury to any of these muscles brings about a disability which may involve one or all of these functions. The oblique muscles should not be cut across but split in the direction of their fibers. This preserves the nerve supply and does the least injury to the bloodvessels. The recti muscles may be cut across without serious damage if properly repaired afterward. It is seldom necessary to cut these muscles as they can be retracted outwardly or inwardly as the case may be. If these muscles are split in the direction of their fibers that portion mesial to the incision will atrophy, its nerve supply having been destroyed. Assmays' investigation proved this to be true and I have confirmed his findings. In 3 cases previously operated at other clinics I found at reoperation that mesial portion had disappeared and was replaced by fibrous tissue.

The main source of blood supply to the abdominal wall is from the lower intercostal arteries, the superior epigastric, the deep epigastric and the deep circumflex iliac. The anastomosis is so free that to cut any of them does not bring serious result, but the injury may lead to infection owing to a lessened blood supply.

The motor nerves (Fig. 1) are of the greatest importance. They are eight in number—sixth to twelfth thoracic inclusive, the iliohypogastric and the ilioinguinal. The thoracic nerves are the ones most commonly injured. They run downward and inward between the internal oblique and transversalis muscles, giving off branches to the oblique muscles and terminating in the rectus muscle on its posterior surface near

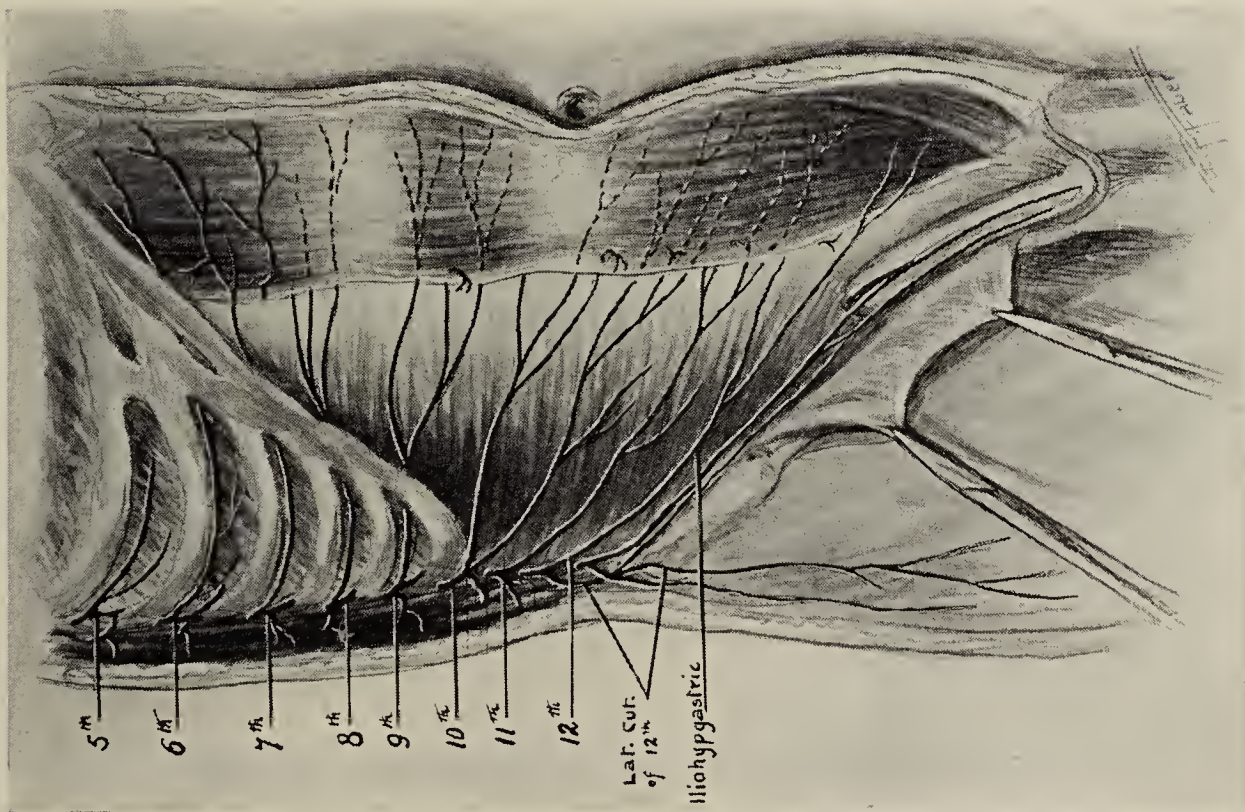


FIG. 1.—Showing nerve supply of abdominal wall.

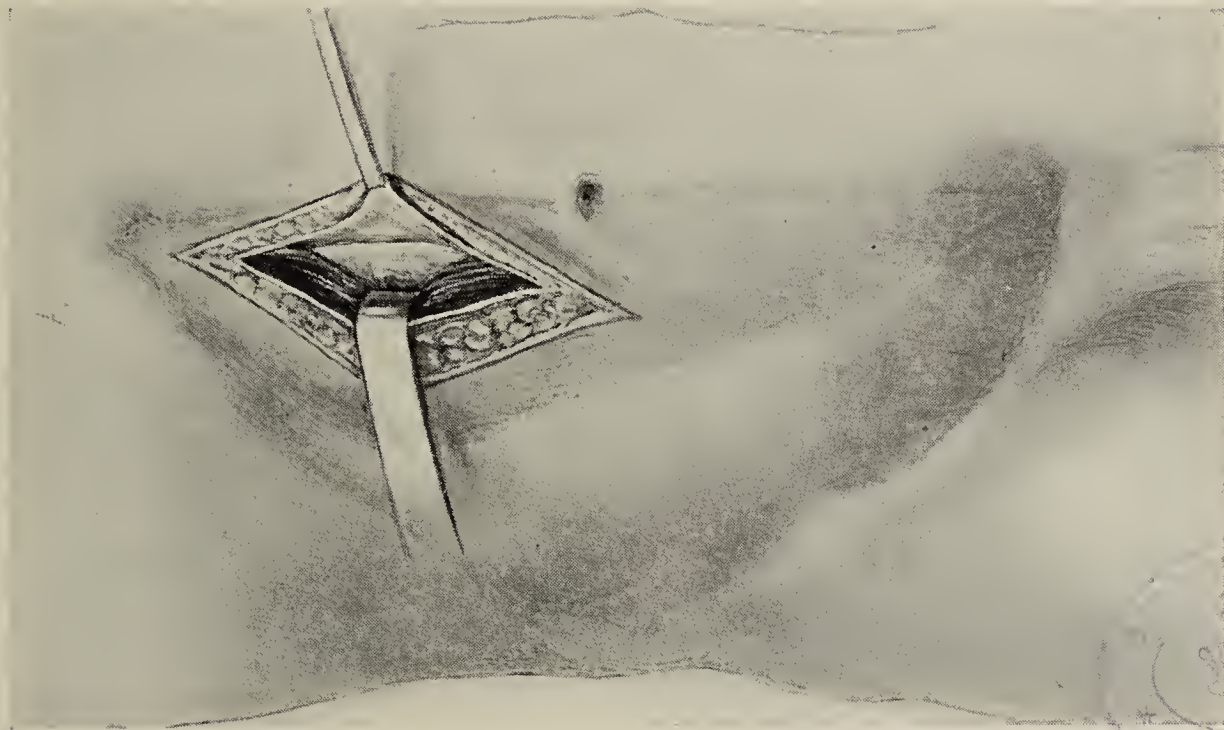


FIG. 2.—Right paramedian epigastric incision, rectus retracted outwardly.

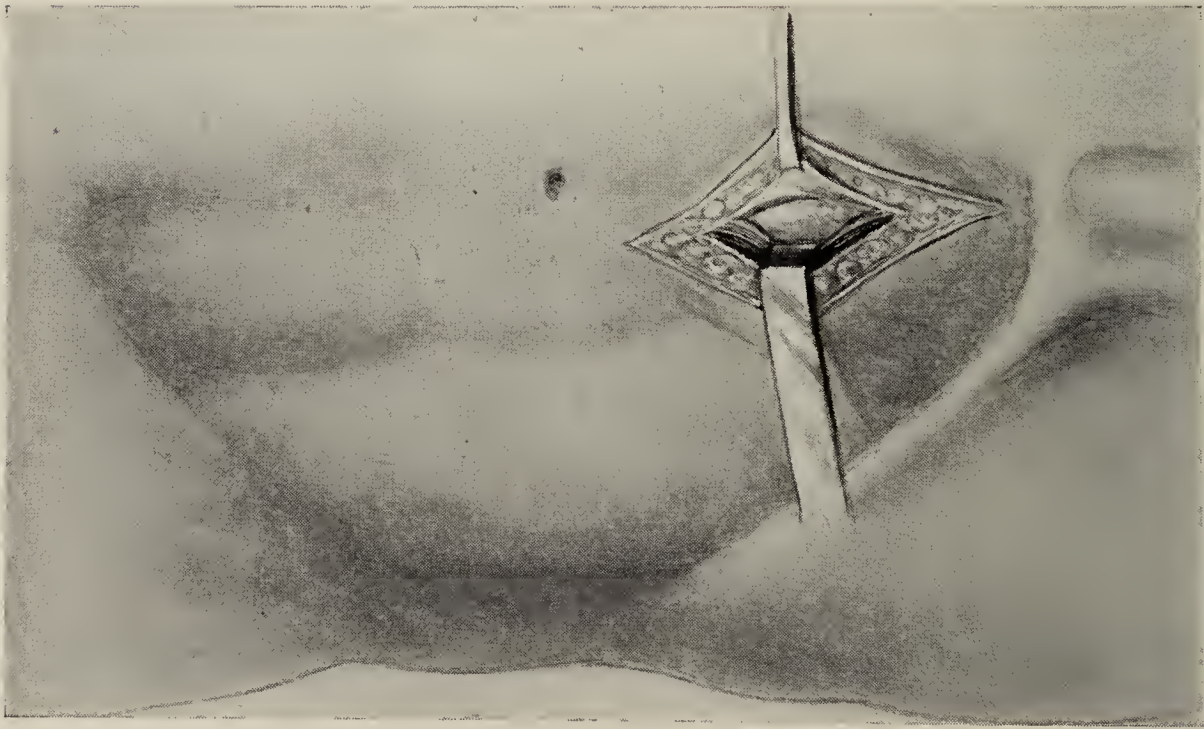


FIG. 3.—Right paramedian hypogastric incision, rectus muscle retracted outwardly.

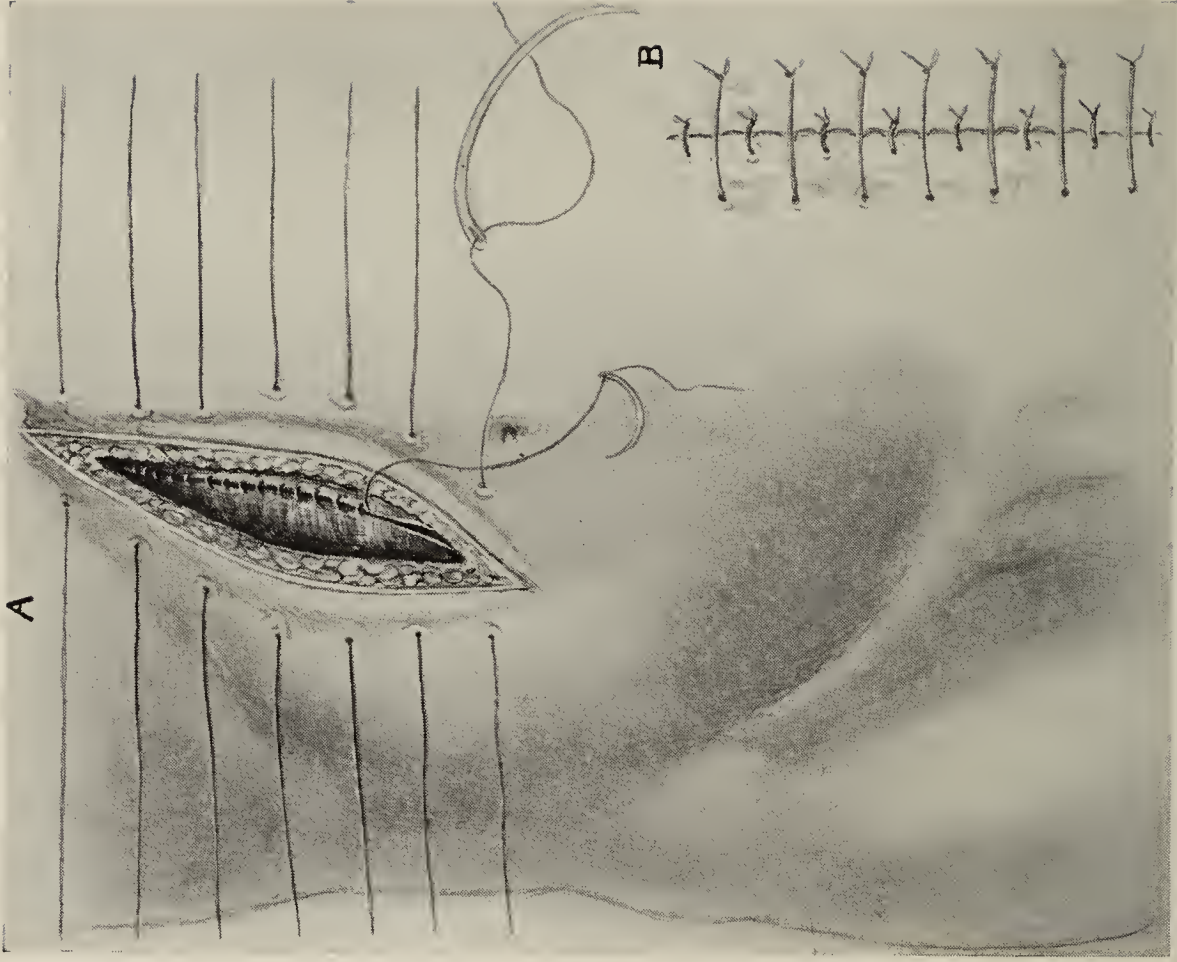


FIG. 4.—A, Right paramedian epigastric incision, oblique. Used for surgery of gall-bladder and bile-ducts. Splint sutures in place. Aponeurosis sutured. B. Splint sutures tied.

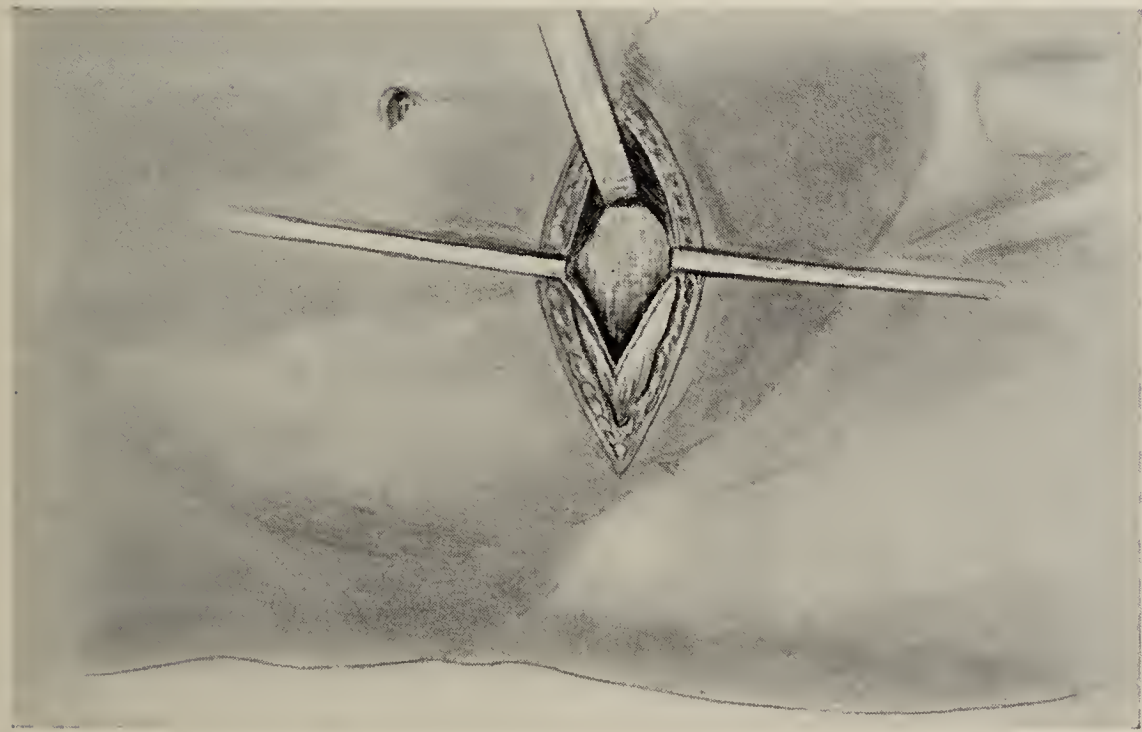


FIG. 5.—Transverse incision of G. G. Davis. Anterior sheath of rectus and aponeurosis of external oblique split. Rectus retracted inwardly.

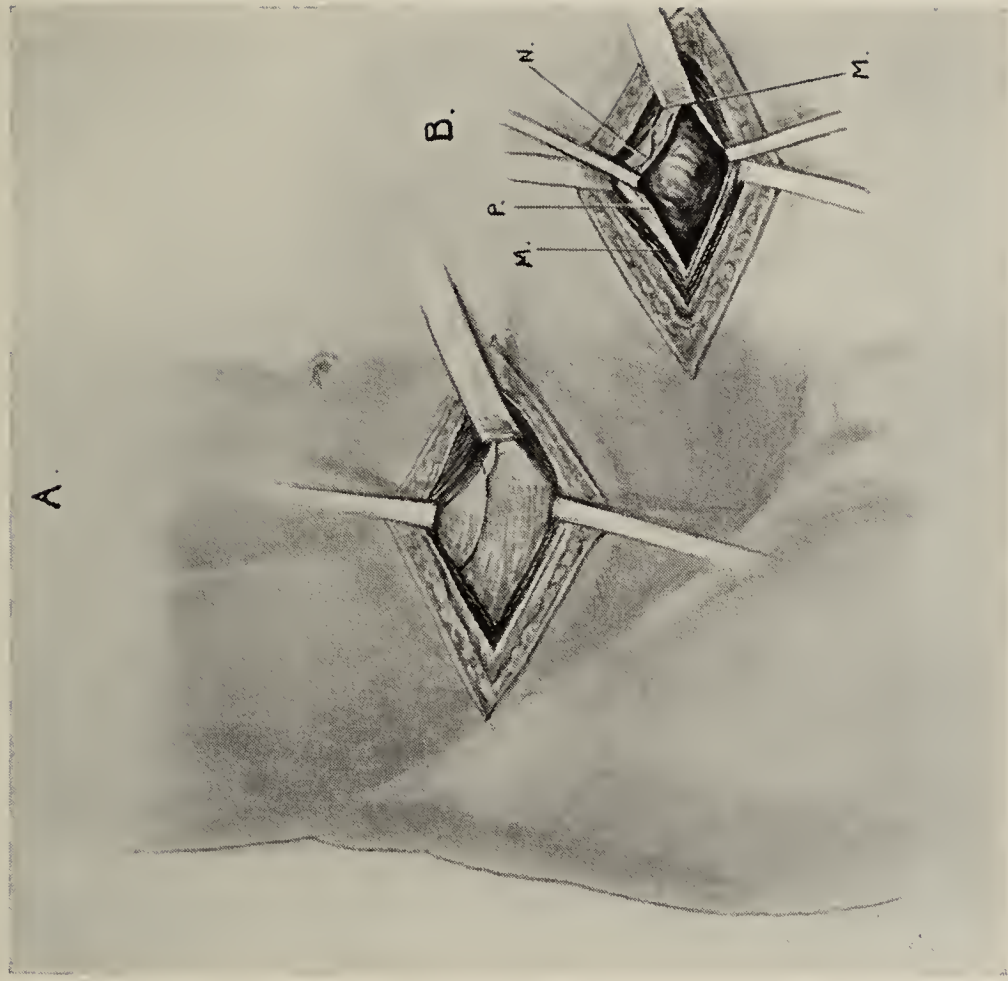


FIG. 6.—A, Internal oblique split exposing twelfth thoracic nerve. B, Nerve pushed aside—peritoneum opened.

the outer border. It can readily be seen that any incision running across these nerves will sever them. The resulting defect is not a hernia but a bulging due to paralysis which cannot be cured by surgery.

The incisions commonly used in the upper abdomen and in the pelvis are through the right or left rectus muscle or through the linea alba. As has been previously stated, an incision which splits the recti muscles produces a permanent defect. Midline incisions are defective also in that they give but one facial plane to suture which is insecure and also leads to a diastasis recti. In the clinic of Doctor Ashhurst we use routinely in the upper abdomen and in the pelvis the right or left paramedian incision (Figs. 2 and 3). After opening the anterior sheath of the rectus the muscle is dissected free along its inner border and retracted outwardly. The posterior sheath and peritoneum are opened beneath. The nerve and blood supply are thus preserved. The aponeurotic layers having been cut in different sagittal planes, the securest closure can be obtained. In operations upon the gall-bladder, instead of using the right rectus, the pararectus, the Kocher, or the Mayo-Robson incision, all of which leave permanent defects, we use a right paramedian oblique incision (Fig. 4). It begins across the midline just below the ensiform, extending downward and outward across the right rectus to just beyond the linea semilunaris at a point below the level of the umbilicus. The anterior sheath of the rectus is opened in the direction of the skin incision, the muscle is dissected free along its inner border, and lifted outwardly, and the posterior sheath and peritoneum opened beneath in a line parallel to the linea alba. This incision gives ample exposure to the fundus of the gall-bladder and an excellent exposure to the bile-ducts which is of much greater importance. Here again the fasciæ are severed in different sagittal planes.

In operations on the appendix there are several incisions commonly used all of which are open to criticism. The gridiron or muscle-splitting incision of McBurney gives a poor exposure and often has to be enlarged. In so doing it ceases to be a muscle-splitting and becomes a muscle-cutting incision. The pararectus or the incision through the linea semilunaris

gives a good exposure, but unless very short it necessarily severs one or more of the thoracic nerves. The same may be said of the incision through the right rectus muscle. The Hancock incision which runs parallel and close to the crest of the ilium above and to Poupart's ligament below, cuts across the bellies of the oblique muscles and often severs the twelfth thoracic and iliohypogastric nerve. We use routinely the transverse incision of G. G. Davis (Figs. 5 and 6). It permits excellent exposure and cuts no muscles or nerves. The incision is so uncommonly used, yet has such obvious advantages, that I will give briefly the technic. It is on a line which extends from the anterior-superior spine of the ilium to the linea alba. The incision is usually 6 to 8 cm. long and centers on the linea semilunaris. It may be made at a higher or lower level than this should the appendix be previously located in other than its usual position. The skin and subcutaneous tissues are cut, exposing the aponeurosis of the external oblique and the anterior sheath of the rectus. These are split in the direction of the skin incision and the rectus retracted inwardly, thus putting on a stretch the internal oblique and transversalis muscles which are split outwardly in the direction of their fibers. The posterior sheath is then split transversely inward as far as needed, and the peritoneum opened for the length of the incision. The twelfth thoracic nerve is sometimes encountered running nearly parallel with the incision, but injury to it is obviated by pushing it to one side. When better exposure is needed the incision may be extended outwardly to the anterior superior spine and inwardly to the linea alba. It is never necessary to cut across the rectus, it can always be retracted medially. At a much higher level this incision may be used for operation upon the gall-bladder, but I do not believe it gives as good an exposure as the one used by us which was previously described. On the left side this transverse incision may be used for splenectomy. I have used it in three such cases. Here, however, it is necessary to cut across the rectus in order to get sufficient exposure.

When drainage is necessary it is best to let the drain emerge near the linea semilunaris rather than through the bellies of the oblique muscles. Muscle tissues held apart by drains tend

to remain so after removal of the drain resulting in hernia. The aponeurosis when separated by drains are drawn together by the action of their muscles when the drains are removed. The several hernias I have had following this incision have been cases in which drainage was through the outer angle.

In making any abdominal incision, the cut should be perfectly clean; the edges of the muscles, whether separated or displaced, must be well defined. Fraying of the muscle edges by manipulation is to be avoided, and this can be done by first making the incision of sufficient length, and second, by protection of the wound edges. The length of the incision means nothing if made in the proper way.

To secure proper healing of wounds great care must be taken in closure. The peritoneum and its adjacent fascia are drawn together and sutured with a continuous catgut suture, interrupted sutures if there is much tension, so applied as to evert the edges, thus bringing serous surface to serous surface. This insures prompt union and prevents adhesions between scar and viscera. In all except the transverse incision interrupted sutures of a nonabsorbable material, preferably heavy silkworm gut, are placed about 2 cm. apart. These sutures embrace all layers down to the peritoneum. The aponeurotic layer is then sutured with chromic gut. The splint sutures are now tied firmly but not tightly. If too tight they defeat their purpose by strangulating the tissues.

It is customary in some clinics for the operator to push his table aside and have the resident close the abdominal wound while he begins another case. This is bad practice. He who begins an operation should finish it. It is safer for the patient, and should something go wrong, the responsibility can be placed.

Not infrequently one hears of or sees reported cases of breaking down of an abdominal wound with evisceration. It is said that it happens to us all at some time during a surgical career, yet I am of the opinion that it is nearly always preventable. The causes of this calamity may be mentioned in the order of their importance. Faulty making and closure of an incision, constitutional diseases with loss of healing power of tissues, infection, and last, the too early removal of splint

sutures. These should rarely be removed before the tenth day; usually it is better for them to remain in place twelve days or two weeks.

In conclusion, to secure prompt healing and to leave the abdominal wall free of defects, we must have:

1. Due regard for structures of the abdominal wall.
2. Clean-cut incisions without trauma to its tissues.
3. Perfect asepsis.
4. Secure and accurate suturing of the wound in layers.
5. Not too early removal of the splint sutures.

THE PHYSIOLOGY OF THE GALL-BLADDER

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THE physiology of the gall-bladder is definitely and intimately associated with and dependent, to a considerable degree, upon its embryology and anatomy. Indeed, I believe we too frequently fail to consider the importance not only of the embryology and anatomy, but also the physiology of the remainder of the biliary system, the duodenum and the surrounding structures in their significant relations and bearing on the physiology of the gall-bladder. In other words, in morbid conditions of the gall-bladder the term "gall-bladder disease" obscures the picture, and the gall-bladder assumes a solitary stellar position and prominence, with the result that very little consideration is given to the remainder of the biliary system—the liver, the duodenum, the pancreas and the related organs—either in treatment, be it medical or surgical, or in prognostic import.

Embryologically,¹³ in the third week of fetal life, in the floor of the posterior portion of the foregut a thickened area develops, later forming a pouch consisting of an anterior hepatic and a posterior cystic portion. The latter gives rise to the common bile duct, the gall-bladder and the cystic ducts. Probably the major hepatic ducts with the minor hepatic ducts develop from the anterior portion, as does the liver parenchyma. During fetal life the gall-bladder is more or less buried in liver substance and is small in proportion. During infancy it grows rapidly and the adult relation is established by the end of the second year.

Anatomically,⁶ consisting of fundus, body and neck, the gall-bladder lies in the gall-bladder fissure on the under surface of the liver. It is 7.5 cm. long and 2.5 cm. in diameter. It holds about $1\frac{1}{2}$ ounces. It is attached rather loosely to the

liver by connective tissue and the peritoneum. It rests on the transverse colon and the first portion of the duodenum below. The neck of the gall-bladder continues as the cystic duct, both containing constrictions of mucosa, namely, the spiral valves of Heister, which are well marked in early life. Later, the spiral ridge atrophies and is broken up at many places, leaving a detached fold with semilunar outline. The common duct is formed by a union of the cystic and the hepatic ducts at the edge of the portal fissure. It passes downward between the folds of the lesser omentum, in front of the foramen of Winslow, behind the first portion of the duodenum, then between the pancreas and inner wall of second portion of the duodenum (usually here the duct is completely surrounded by pancreatic tissue). On reaching the middle of the second portion of the duodenum, it pierces the inner wall obliquely, expanding into the ampulla of Vater and receiving the pancreatic duct, the duct of Wirsung.

The structure of the gall-bladder consists of three coats⁴—the mucosa, the fibromuscular coat and the serosa. The mucosa is raised into folds bounding polygonal spaces and thereby presents a honeycombed appearance. It is lined with columnar epithelium containing a few tubular mucous glands and lymph nodules. It is limited externally by a poorly developed muscularis mucosæ. In the region of the neck of the gall-bladder the mucous membrane forms valve-like folds projecting into the interior. This layer contains an anastomosis of bloodvessels and a lymphatic plexus.

The fibromuscular coat consists of interlacing bundles, indefinitely arranged, of fibrous tissue, and smooth muscle running longitudinally and obliquely. Here are the chief bloodvessels and lymphatic vessels and also a nerve plexus.

The serosa is formed by the peritoneum and is found only on the sides and lower surface. The ducts are fibromuscular structures lined with mucous membrane.

Historically, the function of the gall-bladder was held by Draper (1858) to be that of a reservoir for the storage of bile, where it was concentrated by the removal of a part of its water, undergoing at the same time a change in color. Dalton (1882) believed that the bile is stored in the gall-bladder during

intervals of digestion and passes into the duodenum in largest quantity after eating food. Foster (1889) states that bile is affected by its sojourn in the reservoir of the gall-bladder, becoming green and thicker because of mucus. When the acid contents of the stomach are poured over the orifice of the biliary duct a gush of bile takes place, but alkaline liquids have little or no effect. Foster also believes that the discharge of bile is due to the contraction of the gall-bladder and the associated relaxation of the sphincter of the orifice. He infers that both acts are probably reflex in character, but that the details of the mechanism have not been worked out. Waller (1893) declares that the expulsion of the bile from the gall-bladder occurs in response to the acid-chyme stimulation of the duodenum. Touching the duodenal opening with a glass rod dipped in dilute acid causes a gush of bile. Howell (1900) says that bile is stored in the gall-bladder, and at intervals during digestion is forced into the duodenum by gall-bladder contraction and by the relaxation of the sphincter-like muscle at the orifice, and that these acts are controlled by a nervous reflex occasioned by the passage of acid chyme into the duodenum. Fats and digested protein products (peptones and proteoses) are the most efficient stimuli, while acids, alkalis and starch are ineffective. Starling (1907) states that the secretion of bile is continuous, being stored in the gall-bladder during fasting and discharged by the gall-bladder contraction when acid chyme passes the orifice of the common duct; that the bile secretion is quickened by the injection of secretin into the blood-stream, and that the bile during its stay in the gall-bladder loses its water and acquires mucin, secreted by its mucosa. Martin and Weymouth (1928) believe that the gall-bladder is a temporary reservoir from which bile may be ejected intermittently, plus the remarkable power of concentrating the bile by absorbing water with mucin, and perhaps cholesterol added by the mucosa.

The more recent investigators of the function of the gall-bladder are divided into two groups: One group holds that this vesicle is a reservoir for the storage and concentration of the bile, which later is to be delivered into the duodenum following the ingestion of certain types of food to meet the

demands of intestinal digestion for an additional quantity of bile. The other group contends that the bile enters the gall-bladder through the cystic duct and never leaves the gall-bladder by way of the cystic duct.

That the gall-bladder is a reservoir for the storage and concentration of bile, and later its delivery into the duodenum, is the conclusion of the majority of the investigators in this field. The mechanism for the delivery of the bile from the gall-bladder into the duodenum has long been an unsettled problem, but with the advent of cholecystographic means of studying the mechanism of the biliary vesicle much definite evidence has been collected.

Both groups of investigators agree that the gall-bladder is a reservoir for the reception and storage of bile. In this paper I shall not discuss in detail the mechanism of filling the reservoir and the factors concerned therein.

The delivery of bile from the gall-bladder involves an expulsive force, with the relaxation of the orificial or duodenal musculature. This expulsive force may be extrinsic or intrinsic in character. If intrinsic, it may be active by virtue of muscle contraction or passive by virtue of elasticity.

The extrinsic factors to be considered are: (1) The "respiratory squeeze" of the diaphragm and the liver, advanced by Winklestein; (2) the sphincter of Oddi; (3) siphonage; (4) suction exerted by duodenal peristalsis; (5) the secretory pressure of the liver. Whitaker (1926), by experimental work on dogs, disproved the action of the respiration as a factor in the emptying of the gall-bladder. In addition, during the act of respiration there is a reciprocal innervation between the diaphragm and the abdominal muscles, so that, while the diaphragm contracts, the abdominal muscles relax, thus effectively relieving any counterpressure from below. Cannon has emphasized that the abdominal cavity is a closed chamber; that the specific gravity of its contents is fairly uniform, and that hydrostatic conditions are approximately the same as those in such a chamber, that is, pressure applied to one surface by the piston-like action of the diaphragm would be transmitted equally in all directions throughout the abdominal contents. Upon the descent of the diaphragm the pressure in

the cystic duct, the common duct and the duodenum, especially, since these are collapsible, would equal that in the gall-bladder, and effectively prevent the emptying of the gall-bladder. Higgins and Mann (1926), in their work on fishes, guinea-pigs and dogs, discredited the influence of the changes in intraabdominal pressure as a factor in gall-bladder evacuation. In addition to this, these investigators believe that the secretory pressure of the liver is of little significance in the emptying of the biliary vesicle.

Whitaker (1926) observed under the fluoroscope no extrusion of the gall-bladder contents during inspiration or struggling of the dog after the sphincter of Oddi had been cut. Higgins and Mann (1926)—by the ligation of the hepatic ducts in a dog, thereby eliminating the secretory pressure of the liver; by inserting a catheter into the common duct, thereby ruling out the inhibiting effects of the sphincter of Oddi; by allowing the abdomen to remain open, certainly minimizing the force exerted by the act of inspiration—found that hepatic secretory pressure, the effect of the sphincter of Oddi, and respiratory influences, were of no consequence in emptying the gall-bladder.

Siphonage as a factor cannot exist because the vesicle and ducts are collapsible and enclosed in a hydraulic chamber whose contents are of fairly uniform specific gravity; therefore, none of the required physical conditions of a siphon are present.

Suction as a result of duodenal peristalsis is unreasonable because the ducts are collapsible. Whitaker (1926) fails to find any effect of duodenal peristalsis on the emptying of the gall-bladder. Boyden and Birch (1926), in their experiments on cats, did not observe the sucking action of peristalsis in emptying the gall-bladder. In addition, in their studies of human beings they found no evidence that the mechanical passage of food through the intestinal tract has any influence on the emptying of the gall-bladder.

Regarding the sphincter mechanism, so called, at the duodenal orifice, Berg and Jobling (1926) divided and transplanted the common duct in two dogs, and concluded that normal cholecystograms may be obtained. This excludes the reciprocal relation between the sphincter of Oddi and the gall-bladder. Whitaker (1926) believes that the rôle of the

sphincter of Oddi is to allow the gall-bladder to fill during the intervals between periods of digestion and that the gall-bladder will not empty when the sphincter is cut unless there is resort to feeding. Higgins and Mann (1926) concluded that the sphincter of the common duct is not a factor in the emptying of the gall-bladder, except that its relaxation permits the bile to pass into the duodenum by virtue of vesicle contraction. Mann and Higgins (1927) observed evidence of a strong sphincteric mechanism at the lower end of the choledochus wholly apart from the gastrointestinal tract.

Thus eliminating the effects of extrinsic factors upon the emptying of the gall-bladder, the intrinsic forces must account for its expulsive force.

In 1923 Boyden observed that in cats the gall-bladder is almost completely collapsed a few hours after a meal of egg-yolk and cream; that a lesser degree of emptying occurred following ingestion of lean meat, and very little, if at all, after carbohydrates. About the same time, Whitaker and his co-workers, using the cholecystographic method in human beings and dogs, found that the ingestion of food rich in fat was nearly always followed by a decrease in the size of the shadow of the gall-bladder, with its eventual disappearance in three to six hours. Whitaker (1926), after investigating by cholecystographic methods, following (1) the intravenous injection of opaque halogen compounds of phenolphthalein, and (2) by direct injection of iodized oil into the gall-bladder, after laparotomy, came to the following conclusions: (*a*) that the gall-bladder empties its contents into the duodenum during the digestion of fats; (*b*) the emptying of the gall-bladder is produced by its muscular coat; (*c*) the emptying probably does not depend upon a reflex nervous mechanism involving extrinsic nerves, since vagal stimulation has no effect and denervated gall-bladders empty normally. In addition, he believes that there is no direct evidence of a hormone causing the expulsive action of the gall-bladder, although it invariably depends upon the digestion and absorption of proteins or fats, especially the latter. Furthermore, he states that the wall of the organ contains a large amount of elastic tissue, which may reinforce the action of the smooth muscle if the gall-bladder happens to

be distended when it starts to empty. It is probable, in balancing this pressure exerted by this elastic tissue, that the sphincter of Oddi is effective. Higgins and Mann (1926), using cholecystographic and operative procedures on fishes, amphibia, birds, dogs and guinea-pigs, concluded that the gall-bladder empties through the cystic duct and that it empties by the contraction of its own musculature. Boyden (1926), while studying the contraction of the human gall-bladder, found in a series of cases that the contraction rate was faster in females than in males. Silverman, Denis and Weinberg (1929), investigating the relation of gall-bladder emptying to the ingestion of fats, found that already-digested fats do not affect the gall-bladder reflex and that, therefore, there is some relationship between the gall-bladder emptying and the intestinal digestion of fats. Krause and Whitaker (1928), in studying the effect of different foods upon the emptying of the gall-bladder, found that fats and fatty acids are the most active foods in the emptying of the gall-bladder; that pure carbohydrates are practically ineffective, and that proteins are but slightly effective in emptying the gall-bladder. Lueth, Orndorff and Ivy, studying the effect of histamine on the gall-bladder contraction, found that it occasionally causes the gall-bladder to evacuate, but does not offer the possibility of being a gall-bladder test.

Ivy and Goldberg (1928), using secretin which was highly purified vasodilation-free, and having no objective toxic effect on anesthetized and unanesthetized animals, found that this extract of upper intestinal mucosa following intravenous injection produces contraction and evacuation of the gall-bladder, and that cross-circulation experiments show that when acid is injected into the duodenum something gets into the blood-stream causing the gall-bladder to contract. In view of this, these investigators believe a hormone mechanism causes the contraction and evacuation of this viscus, and they propose the name of cholecystokinine for this hormone. Further they found that the injection of tenth-normal hydrochloric acid, butter, digested egg-yolk, cream and olive oil, 0.5 per cent butyric acid and 5 per cent soap solution into the duodenum of a dog caused the gall-bladder to contract. Undi-

gested olive oil, cream and egg-yolk were ineffectual. Scott and Whitaker (1928), in a series of cholecystographic studies, conclude that the musculature of the gall-bladder contracts and expels the contents of the vesicle following a feeding of fat, and that various conditions affecting the tonus of smooth muscle must be considered in the interpretation of the motor phase of any cholecystographic studies. Lyon (1929), in a study on the cholecystographic evidence of biliary drainage, concludes that the gall-bladder empties its contents by means of its intrinsic musculature, and that the intraduodenal stimulation with magnesium sulphate, peptone and olive oil secures specimens of vesicle and liver bile.

Burget (1926), in contradistinction to the previous view, believes that the smooth muscle tissue, as shown by Hendrickson in the gall-bladder of man and the dog, is too sparse and is not arranged in coats as we would expect in an organ possessing powers of contraction. He suggests that the bile flow observed by the previous investigators as due to gall-bladder contraction might better be interpreted as due to peristalsis and the lowered tonus of the duodenum. Further, that the rise in pressure, intravesicular, reported by others, is the result of a strong tonus rhythm, and it is doubtful if they could play a part in the expulsion of the bile, since they disappear when the pressure in the gall-bladder rises to the height necessary to overcome the resistance of the duodenum.

Halpert and Hancke, working on the function of the gall-bladder in rabbits by injecting methylene blue intravenously and by oral administration, conclude that methylene blue appears in the bile after intravenous or oral administration; that the methylene blue appears in the bile collected from the ductus choledochus within a few minutes after intravenous injection; that the bile removed from the gall-bladder three hours after injection contains two to twenty-two times as much of the dye as the last sample collected from the ductus choledochus; that the concentration of dye in the bile removed from the gall-bladder is at times much higher than the highest concentration ever reached in the bile coming from the liver; that the gall-bladder does not secrete the dye (all the methylene blue reaching the gall-bladder by way of the cystic duct);

that pressure exerted on the liver during inspiration is the main force driving bile into the gall-bladder; that the bile collected from the ductus choledochus between twelve and seventy-two hours after feeding the dye contained little or no methylene blue; that the gall-bladder resorbs methylene blue and its leuko forms much more slowly than it resorbs bile, and that bile does not leave the vesicle, under ordinary conditions, by the cystic duct.

Sweet and Halpert look upon the gall-bladder as a reservoir for bile, with resorption *in toto* taking place by means of the mucosa, the constituents being returned to the general circulation by way of the veins and lymphatics. They believe that the anatomic arrangement about the neck of the vesicle and in the cystic duct, regulating, as it does, the inflow, and hindering or preventing the outflow, shows a wide enough range of variations from perfect or nearly perfect competency to total derangement, mainly in conditions of biliary stasis, to account for a great deal of the experimental results of the observers who believe that the vesicle is a reservoir for storage and, later, for expulsion of bile by the contraction of its intrinsic musculature.

In conclusion, the greater mass of evidence and the prevalent opinion among the majority of the investigators in the field of gall-bladder physiology point to this viscus as being a reservoir for the storage and concentration of bile, which is later to be expelled from it by the contraction of its own intrinsic musculature in response to the presence of gastric digestive products in the duodenum, especially fats; the contraction stimulus being, in all probability, humoral in character, although a neural or humeroneural regulatory mechanism (as yet not found) must not be overlooked as a possible factor in biliary vesicle physiology. Furthermore, the gall-bladder bile undergoes certain changes—the mucosa adding to and taking from it certain constituents—thereby so-called concentration taking place. Regarding these changes, biochemistry has told us little.

Another significant factor is the rôle played by the condition of the duodenal musculature and the orificial mechanism at the outlet of the common duct, which by its opening or closing

affects not only the entrance of bile into the duodenum but also the entrance of the pancreatic secretion. Finally, the interdependence and interrelation of all these parts signifies the importance of considering the biliary tract as a whole.

The physiology of the biliary system in its entirety, considering the function of the gall-bladder as a component part of that system, is of paramount importance if we are to interpret intelligently the normal physiology and treat logically and successfully the morbid physiology, not of the gall-bladder along, but of the entire intrahepatic and extrahepatic biliary system.

In summarizing the foregoing results of various investigators in the field of gall-bladder physiology, the writer concludes that:

1. The gall-bladder is a fibromuscular organ which contracts by virtue of its own intrinsic musculature.
2. The contents of the vesicle are expelled through the cystic duct into the common duct and thence into the duodenum.
3. The stimulus for contraction is in all probability humoral or hormonal in character.
4. The gall-bladder possesses the power of adding to and taking from the bile certain constituents.
5. The sphincter mechanism at the duodenal orifice of the common duct, in conjunction with the condition of the duodenal musculature, influences greatly the filling of the biliary vesicle, and, after contraction, inhibits or enhances the flow of bile into the duodenum from the common duct.

BIBLIOGRAPHY

1. Berg, B. N. and Jobling, J. W.: *Proc. Soc. Exper. Biol. and Med.*, 1926-1927, xxiv.
2. Boyden, E. A. and Birch, C. L.: *Proc. Soc. Exper. Biol. and Med.*, 1926-1927, xxiv.
3. Boyden, E. A.: *Proc. Soc. Exper. Biol. and Med.*, 1926-1927, xxiv.
4. Burget, G. E.: *Proc. Soc. Exper. Biol. and Med.*, 1926-1927, xxiv.
5. Dalton, T. C.: *Human Physiology*, Philadelphia, 1882.
6. Davis, G. G.: *Applied Anatomy*, Philadelphia, 1924, pp. 424-426.
7. Draper, T. C.: *Human Physiology*, Philadelphia, 1858.
8. Foster F.: *A Text-book of Physiology*, New York, 1889, p. 421.
9. Halpert, B. and Hanke, M. T.: *Am. Jour. Physiol.*, 1929, lxxxviii, 307.
10. Higgins, G. M. and Mann, F. C.: *Am. Jour. Physiol.*, 1926, lxxviii, 339.

11. Howell, W. H.: Text-book of Physiology, Philadelphia, 1900, p. 248.
12. Ivey, A. C. and Goldberg, E.: Am. Jour. Physiol., 1928, lxxxvi, 599.
13. Jackson, C. M.: Morris' Human Anatomy, Philadelphia, 1921, xlv, 1214.
14. Krause, W. F. and Whitaker, L. R.: Am. Jour. Physiol., 1928, lxxxvii, 172.
15. Lueth, H. C. and Orndorff, B. H.: Proc. Soc. Exper. Biol. and Med., 1929, xxvi, 311, 312.
16. Lyon, B. B.: Arch. Int. Med., 1929, xliii, 147.
17. Mann, F. C. and Higgins, G. M.: Proc. Soc. Exper. Biol. and Med., 1926-1927, xxiv.
18. Martin, E. and Weymouth, F. W.: Elements of Physiology, Philadelphia, 1928.
19. Scott, W. J. M. and Whitaker, L. R.: Jour. Am. Med. Assn., 1928, xci, 9.
20. Silverman, D. N., Denis, W. and Weinberger, H. L.: Am. Jour. Med. Sci., 1929, clxxvii, 384.
21. Starling, E. H.: Elements of Human Physiology, Chicago, 1907, p. 240.
22. Waller, A. D.: An Introduction to Human Physiology, London, 1893.
23. Whitaker, L. R.: Am. Jour. Physiol., 1926, lxxviii, 411-435.

SEVENTY-FIVE YEARS OF GALL-BLADDER SURGERY

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EARLY literature contains only sporadic references to the gall-bladder or gall-stones, while any mention of gall-bladder surgery mainly concerns disputed questions of the anatomy and physiology of the organ, the main point of controversy, which was actively debated from the fifteenth to the eighteenth century, being whether the gall-bladder receives its bile by way of the cystic duct or directly by way of the hepaticocystic ductlets; in the latter circumstances the cystic duct would represent merely an excretory duct. It was in this connection that the first experimental removal of the gall-bladder of the living dog, by the Italian Zambecari, in 1630, proved that the gall-bladder is not essential to life. To this, the first epoch in gall-bladder surgery, belong also the first intimations of the chemistry of gall-stones by Ettmueller, who, with his pronouncement that gall-stones cannot be dissolved by medication, and even if recovery seems to take place in a given case of cholelithiasis, recurrences will surely follow and eventually lead to death, started the still-active controversy of medical *versus* surgical treatment of gall-stone disease.

The first deliberately planned operation on the biliary system is generally credited to the Frenchman, Louis Petit (1743). His indication for operation was limited to cases in which a diagnosis of adhesions had been made. That is to say, cases which presented a tumor or an enlarged, adherent gall-bladder—one that could not be moved from side to side—or cases in which the organ had perforated, as evidenced by an inflammatory swelling of the belly wall. The operation naturally was limited to the area of adhesions, and consisted either of puncture of the bile-distended gall-bladder or incision into the calculous gall-bladder.

This epoch was followed by much speculation and more or less activity in animal experimentation, although the bold idea of a cholecystectomy was entertained by one Herlin as early as 1767. It goes without saying, however, that the success of such a procedure awaited the discovery of anesthesia and antisepsis. It is not surprising, therefore, that Herlin's suggestion was doomed to oblivion until the birth of modern surgery.

Even for almost two decades after the introduction of anesthesia little is said of surgical treatment of disease of the gall-bladder. While the recognition of certain digestive disorders, especially the violent colicky pain of the calculous disease, made the recognition of the seat of the disorder easy, it was not until the introduction of the microscope and the theory of bacterial invasion became prevalent as the cause of many abdominal disorders, that the rôle of infection as the main factor held sway. Treatment of the disorder known as gall-stone disease for a long time was medical and dietetic. Cholagogues were freely administered with the idea of washing out the toxins by way of the bowels, and the diet regulated to aid digestion. Spa treatment, a happy combination of the two, was a popular form of therapy as travel became more common through the introduction of railways and of steamships for ocean travel.

In the course of time, with the development of chemotherapy and bacteriology, treatment was directed to disinfecting the bile tract, as well as to stimulating the secretion of bile and also to supplying deficiencies in the composition of the bile. These measures, however, soon proved to be only palliative, since they evidently failed to remove the toxic bile from the system promptly enough to prevent its resorption by the lymph circulation or the blood-stream. More radical treatment, therefore, seemed to be demanded. Emboldened by the gradually increasing field of surgery as anesthesia and asepsis were beginning their beneficent reign, it was only natural that the surgeon should attempt the cure which medical measures were evidently powerless to effect.

I have already briefly indicated the first steps in this direction, which remained sporadic and more or less empirical until

the year 1867. At this time Bobbs, of Indianapolis, performed the first cholecystostomy, although he had mistaken the swollen gall-bladder for an ovarian tumor. At any rate the possibility of surgical drainage of the gall-bladder was demonstrated. Strange to say, however, it took another decade before a deliberately planned cholecystostomy is reported, the operation having been done by that pioneer in abdominal surgery, Marion Sims.

Bobbs, in his operation, drew the gall-bladder out of the wound, incised it, and after removing the calculi, closed the gall-bladder incision. Sims, in describing his operation, stresses the value of antiseptic precautions—the carbolic spray and antiseptically prepared instruments—without which, he declares, the procedure would have been impossible. He made an incision over the most prominent part of the tumor, through which he thrust a large trocar and evacuated about 24 ounces of a dark-brown fluid which he supposed to be bile. After the cyst was evacuated he hooked it up with a tenaculum and pulled it out into the wound for about 2 inches; examination revealed it to be the gall-bladder. He then made a 2-inch incision into the gall-bladder and thoroughly cleansed it out with sponge probangs, obtaining mucoid bile much thicker than that at first obtained through the trocar, and then gradually removed the stones, about sixty in number. He next sewed the open end of the gall-bladder to the upper angle of the abdominal incision, thus establishing a fistulous outlet. At this point he confesses having made the mistake of amputating a projecting portion of the gall-bladder itself. Its walls were thickened and there was considerable bleeding. “The puckered mouth of the amputated cyst was then crowded into the upper angle of the abdominal incision,” and secured with sutures passed through the entire thickness of the abdominal walls, as well as the peritoneum, after which the abdominal incision was closed without drainage. The patient did well until the sixth day, when the discharge on the dressings was seen to be mixed with blood; this was soon followed by frank hemorrhage per mouth, and death ensued on the eighth day. Postmortem revealed entire absence of peritonitis—“a triumph of Listerism,” to use Sims’ own words. “Death,” he declares,

“was due to occlusion of the bile ducts from transudation of blood from the mucous surfaces; that is to say, passive internal hemorrhage, the result of the poisonous effects of the biliary salts on the blood”—the patient’s blood having been impoverished by the prolonged toxic action of the bile. In spite of its unfavorable outcome, the clinical history of the case and postmortem findings established the safety of the operation *per se*. Furthermore, to Sims, it provided a good argument for early intervention before the toxicity of the patient reduces the chances of success: an argument that has lost none of its validity even in our own day, in spite of the advances in our knowledge of the nature of the disease and our improved operative technique. Sims regretted having removed a piece of the gall-bladder, a step which he decided to omit in the future, and he predicted for this surgical measure the same impunity as obtained for exploratory laparotomy for other abdominal conditions which was then rapidly coming into vogue and which Listerism had made feasible, safe and justifiable. After this, numerous similar operations for gall-stones were reported in quick succession by Keen, Lawson Tait, Rosenbach, Ronsahoff and others. It remained, however, for Carl Langenbuch, in the year 1882, to inaugurate the third era in gall-bladder surgery with the introduction of the radical removal of the gall-bladder itself in the treatment of gall-stones.

Langenbuch was led to consider the feasibility of the operation by the prolonged suffering and repeated attacks of gall-stone colic, endured by a patient, leading to death with every evidence of metastatic ulceration within the abdomen, the direct result of the gall-stone disease.

That the gall-bladder is not essential to the life of the mammal was evident to him not only by the absence of the organ in such animals as the elephant and the horse, but also by its congenital absence in the human subject, as observed postmortem, without having affected the well-being of the individual concerned. This fact encouraged Langenbuch to attempt its removal as a therapeutic measure. After repeated experiments on the cadaver, he finally arrived at the conclusion that of all abdominal operations, extirpation of the gall-bladder, with previous ligation of the cystic duct, was perhaps

the least radical. His technique is as follows: Two incisions are made forming the letter T, each incision being about 10 to 15 cm. in length, along the lateral border of the right rectus muscle. The gall-bladder presents with its attachments to the under surface of the liver and its tip free. With a flat sponge introduced into the wound, the colon and small intestine below the abdominal incision are pushed downward. The right lobe of the liver is seen inclined forward; raising this puts the hepaticoduodenal ligament on the stretch, so that it protrudes and can be grasped with the fingers of the left hand. This fold contains the large biliary ducts and the portal vessels. In order to get at the cystic duct at its rather isolated location farthest to the right, the gall-bladder must be dissected free. The organ gradually diminishes in size and finally merges with the cystic duct. The latter is ligated, at a point about 2 cm. from the gall-bladder, with silk thread, catgut being unsuitable, since the object is to obtain permanent closure of the duct. The gall-bladder is then carefully freed from its bed, after which the cystic duct is severed at the proximal side of the ligature. Should the gall-bladder be distended with bile, before removing it, it can be emptied by aspiration in order to prevent soiling the wound. Every precaution must be taken to avoid injuring the liver. Closure of the wound finishes the procedure, in which, with the exception of the right colic flexure, scarcely any of the intestinal tract is exposed.

Langenbuch soon had an opportunity to put his studies to a test. On July 15, 1882, he performed the operation on a male, aged forty-three years, and cured his patient, thus establishing cholecystectomy as a practical measure from a physiological, technical and clinical point of view. Not only did this achievement usher in a new phase of gall-bladder therapy, but it opened up the way for an overwhelming amount of speculation and research into the pathogenesis of disease of the gall-bladder, especially cholelithiasis, the end of which is not yet in sight. There are few subjects which present a richer, more varied and, one might say, a more confusing literature.

That the primary agent in disease of the biliary apparatus (neoplasms excepted) is infection, soon became a generally

accepted fact, the possible avenues being through the portal circulation, the general circulation, the lymph stream and, by contiguity, with a neighboring infected viscus. Another truth revealed by progressively increasing observations was that the colicky attacks are due not so much to the mechanical factor of the passage of the stones through the narrow cystic duct, but primarily to the inflammation and infection caused by the presence of the stones, and that bile stasis plus an infectious catarrh are prime factors in the formation of stones (Naunyn); a third factor being faulty metabolism (diathesis), which so alters the composition of the bile as to favor the deposit of calculus-forming substances.

From the study afforded by the extirpated gall-bladder there resulted a rather clear picture of the steps in the pathological changes in the diseased organ. Cholelithiasis leads to hypertrophy of the muscularis and an increase in the glandular elements of the glands normally found only in the lower portion of the gall-bladder as well as of the canals of Luschka in which bile may enter and stagnate. A severe inflammation may lead to ulceration and cicatricial formation on the mucosa, which then loses its delicate reticulated structure. Dilatation of the canals of Luschka, with eventual formation of diverticulæ, may follow, permitting small stones to settle in the latter with resultant abscess formation and craterlike ulcers, not due to the stones. Such a condition minimizes, if not totally eliminates, the chance of a cure of the gall-bladder disease. Perforation of such ulcers may lead to pericholecystic or hepatic abscess, the seriousness of which is apparent. In the course of the pathological process there is an increase of the submucous connective tissue which may produce a more or less marked thickening of the gall-bladder. Furthermore, a typical epithelial proliferation, due to the irritating action of the stones, may lead to malignant changes (Koerte). Pathological changes, such as dilatation of the cystic duct, may further complicate conditions in the gall-bladder and eventually affect the common duct, leading to dangerous consequences which by this time are familiar to the profession and even to a large part of the laity. The seriousness of common-duct obstruction and jaundice as concomitant symptoms of calculous disease of the biliary tract is a matter of common knowledge.

Thus, very briefly outlined, some of the facts and possibilities of neglected disease of the gall-bladder present themselves to view and at the same time point out the indications for operation.

With the introduction of cholecystectomy a lively discussion arose on the subject of cholecystostomy *versus* cholecystectomy. At first the radical operation was reserved for the desperate cases, but in the course of time, as individual surgeons became familiar with the radical procedure and gradually improved or modified their technique, they soon found that there was very little difference between the two measures as far as immediate mortality was concerned. That is to say, in the absence of untoward circumstances and in the hands of the experienced surgeon, one operation was as safe as the other. Study of end-results, however, soon caused the pendulum to swing in the direction of radical surgery. In many instances it became evident that conserving the gall-bladder meant conserving it for future attacks of gall-stone disease. It was found that in the majority of instances persistent or recurrent symptoms were due to adhesions and the reformation of gall-stones, so on the principle that "if thy right hand offend, cut it off," cholecystectomy has become the operation of choice, particularly in the hands of the adept surgeon, cholecystostomy being reserved for the desperately sick case or for certain cases of marked obesity or some other condition in which the radical procedure would be contraindicated.

Nevertheless, the conservative operation still is useful, as it permits one or the other of the anastomotic procedures that followed the introduction of the modern era in the history of disease of the gall-bladder and its surgical treatment. The earliest such operation (cholecystenterostomy) is that reported by Von Winniwarter in 1882. His was a cholecystocolostomy, a very complicated procedure that required six sittings for its completion. Naturally it failed to gain popularity; but in a modified form, as a cholecystogastrostomy, it is now occasionally a very useful procedure. The objection to it, observed on reoperated cases, is that the anastomosis does not always remain patulous and the patient's latter state is apt to be as bad if not worse than his prior one.

From the above survey it is seen that the year 1882 is a banner year in the evolution of gall-bladder surgery, which was destined to advance with surprising rapidity. It was not long before the idea of incising the common duct for the removal of a stone was conceived. Langenbuch discussed its possibility in 1884, and it was favorably considered by Parkes as well as by Kocher. But it did not emerge from the theoretical stage until 1890, when it was attempted by Kümmel, and later in the same year successfully performed by Curvoisier. In the early operations on the common duct the attempt was always made to close the incision in the duct after the stone or stones had been removed, but this was soon abandoned and drainage of the duct (choledochostomy) was used in cases of cholangitis or in conjunction with cholecystectomy in the treatment of the calculous gall-bladder.

The question of drainage in cholecystectomy is still a matter of discussion. Personally I may say that I use a drainage (rubber tube) in the subhepatic fossa as a routine procedure in all my cholecystectomies. I believe it a wise measure and have had no reason to change this belief, judging from the results obtained.

An occurrence of some note in the history of gall-bladder disease is the introduction of the Lyon method of nonsurgical drainage of the gall-bladder both as a diagnostic and a therapeutic agent.

In September, 1919, Dr. B. B. Vincent Lyon, of Philadelphia, published the results of experimental studies and clinical observations made by him on the basis of a paper by Dr. S. J. Meltzer, of the Rockefeller Institute of New York, on the "Disturbance of the Law of Contrary Innervation as a Pathogenetic Factor in the Diseases of the Bile Ducts and Gall-bladder." In this paper (in a footnote) Meltzer observes that experimentally a 25 per cent magnesium sulphate solution applied locally to the mucosa of the duodenum causes a local relaxation of the intestinal wall, a reaction that does not occur when the same solution, given by mouth, passes through the stomach before it reaches the intestine. He suggests that in the human subject this direct action on the intestinal mucosa can be accomplished with the use of the duodenal tube, and that it may possibly serve as a test in jaundice and biliary

colic, since it may "relax the sphincter of the common duct, permit the ejection of bile and perhaps even a calculus of moderate size wedged in the duct in front of the papilla of Vater."

Lyon, much impressed with the practical significance of this suggestion, was led to try the method on the human subject, first, with magnesium sulphate and later with other solutions. Arguing from cause to effect, he reasoned that if Meltzer's law of contrary innervation is a sound one, inhibiting the sphincter should stimulate the gall-bladder to contract and empty its contents into the duodenum, and that by means of the duodenal tube the bile could be collected in bottles and studied. After a few attempts the practicability of the method in the diagnosis of biliary disease became evident. In the fasting period the sphincter of the bile duct is closed and there is no bile in the duodenum, so that the introduction of the magnesium sulphate causes the sphincter to open. The first bile obtained would, therefore, be that coming from within the ducts, especially the common duct, the first source of supply. The first bile is of a pure yellow color and syrupy consistency. After a few cubic centimeters have been withdrawn the bile suddenly deepens in color and becomes more viscid. This second bile apparently comes from the gall-bladder. In turn, it is followed by a light lemon-yellowish limpid bile—liver bile. Lyon gives his reasons for believing that the second bile is gall-bladder bile, the most cogent one being that in cholecystectomized patients studied after operation this type of dark bile is not obtained, but the first bile is immediately followed by the third type—the liver bile—which can be collected as rapidly as it is secreted. Furthermore, in most gall-bladderless patients bile continuously enters "the duodenum in the fasting stomach and duodenal state, except in secondary cholangitis with obstructive jaundice, indicating that the duct sphincter is in a state of inhibited tonus, probably permanently so, since the antagonistic or contrary innervation has been cut when the gall-bladder was removed."

Without discussing the merits or demerits of the method, it may suffice to say that it has aroused attention in this country and abroad and has found a place as a method of recognizing gall-bladder derangement perhaps earlier than would other-

wise always be possible, and to some extent also is of therapeutic value in selected cases, particularly where grave contraindications to surgery exist, as well as in the treatment of strictly medical conditions, such as biliary migraine, biliary cirrhosis with or without intestinal autointoxication, catarrhal jaundice, etc.

It is only natural that the question of diagnosis of disease should arouse the interest of the clinician, the surgeon and the pathologist alike. Going back some decades in our narrative, we find that the benefits of Roentgen's brilliant discovery of the x -ray was almost immediately adopted as an important adjunct in the study of gall-stones, their composition, structure and density. The early investigators studied gall-stones outside of the body; the first report on the subject is that by the Frenchmen, Gilbert Fournier and Oudin, followed later by publications by Luis y Jague and Gatezulu, of Spain, and by the Italian, Fornario. These authors all agreed that x -ray visualization of gall-stones depended upon their calcium content, the pure cholesterol stones failing to be visualized. They all expressed the hope that as the apparatus and the methods of study developed, the x -ray would undoubtedly prove a useful method of detecting gall-stones in the living human subject. With the advent of the opaque meal and the fluoroscope this hope was realized, but only to a limited extent—limited because of the uncertainty of its results; that is to say, a negative result could not always be relied upon as evidence of the absence of stone or of pericholecystic or cholecystic disease, the result of infection. Inasmuch as all authorities agree that the seriousness of the condition is due not so much to the gall-stones as to the infection to which they give rise, the importance of a means of detecting early infection becomes self-evident. This means has been made available by the brilliant discovery of cholecystography, in 1923, by Evarts Graham and Warren H. Cole, of St. Louis. In the words of these authors, it (cholecystography) embodies an entirely new original radiological principle, namely, the utilization of the specific functions of a system to engender a difference in density. Cholecystography is, therefore, largely a test of physiological capacity in contradistinction to the almost wholly mechanistic nature of other examinations, such

as the opaque meal, pyelography, etc. On the basis of the work of Abel and Rowntree, that the phenolphthaleins are largely excreted in the bile, and after considerable experimental work of their own, Graham and Cole found when phenolphthalein in combination with iodine or bromine was injected into the veins of animals or man it was "excreted in the bile, and that a sufficient concentration of the halogen was obtained in the gall-bladder to cause a shadow on a film when exposed to the x -ray." This method of visualizing the gall-bladder they termed cholecystography and the exposed films they called cholecystograms. The substance used by them, and mostly by others, is sodium phenoltetraiodophthalein, containing about 60 per cent iodine by weight, the iodine or the bromine being the substance that is opaque to the x -ray. The drug can be administered by mouth or it can be injected into the veins. In the fasting subject its injection intravenously is excreted by the liver and sent to the gall-bladder by the action of the common-duct sphincter. Within the gall-bladder the bile and the iodine undergo concentration, due to the absorptive action of the gall-bladder wall. The process takes about twenty-four hours, and it is during this period that the shadows can be produced on the films by exposing the gall-bladder to the x -ray. The iodine is removed from the gall-bladder in the normal process of emptying itself and the organ is then no longer opaque to the x -ray.

The cycle of filling and emptying of the gall-bladder being thus visualized, its application to the differentiation between the normal and pathological gall-bladder was made possible; the method has also proved of great value in enhancing our knowledge of the physiology of the gall-bladder. In effect its greatest value is a means of early detection of physiological disturbances rather than anatomical changes in the gall-bladder.

This attempt to present some of the salient features in the history of gall-bladder disease as developed in the last seventy-five years, based mainly on the facts as revealed by the development of the surgery of the biliary tract, it is hoped will reëmphasize the importance of surgery in the progressive advancement of diagnosis and therapeusis of disease of the gall-bladder.

TWO CASES OF SUBDIAPHRAGMATIC ABSCESS COMPLICATING APPENDICITIS*

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CASE I.—A boy, G. R. W., aged eight years, was admitted to Dr. Ashhurst's service, in the Episcopal Hospital, on January 16, 1921, at 11 P.M., with a diagnosis by the family physician of empyema. The history of the case is as follows:

The family history was negative; personal history included whooping-cough at seven years and occasional colds; no other diseases, no operations. The chief complaint was cough and dyspnea. The patient was perfectly well, with the exception of a slight cold, until three weeks ago (December 26, 1920) when he was taken with pain in the upper abdomen. This pain came on gradually; was dull in character; not relieved by pressure on the abdomen; not radiating to the back, side or lower abdomen. The next day the pain continued, not severe but annoying. A dose of castor oil was given, but returned, and there developed a slight, hacking, nonproductive cough. The mother thought he had had no fever, and the condition remained about the same, the boy having little appetite, staying in bed part of the time and being up part of the time apparently in this state for the following two weeks.

Twenty days after the onset the condition became much worse. The cough became much more severe, at times nearly causing exhaustion. A yellowish, frothy sputum with a foul odor was coughed up. The patient's breath was also foul-smelling and there was marked dyspnea. He did not complain of abdominal pain. He vomited everything he had eaten, and vomited and gagged after a severe coughing spell. He was seen by the family doctor, who said he thought the child had empyema and should go to the hospital.

On admission the boy was poorly nourished and extremely sick, with rapid and labored respirations, attacks of violent coughing, very foul breath and somewhat cyanotic. There were enlarged

* Reprinted from the New York Medical Journal and Medical Record, 1922, cxvi, 128.

veins under the eyes and over the chest and abdomen. The temperature was 104° F.; respirations, 48 per minute; pulse, 144. The pupils were slightly dilated, reacted to light and accommodation, eyes somewhat sunken.

The tongue was heavily coated and there was a very foul breath. The veins of the neck were prominent, pulsation of the carotids was visible. There was a bulging of the interspaces on the right side; expansion was more marked on the left side. The apex beat was faintly visible and felt in the fifth interspace, $1\frac{1}{2}$ cm. to the left of the nipple line. On percussion the left lung was resonant throughout. The right chest presented dulness, both anteriorly and posteriorly, below the third rib; it was resonant above the third rib. The heart was apparently pushed to the left. There were crackling râles of all varieties throughout the left chest and upper part of the right chest. The breath sounds were much exaggerated over the left side. The right side, above the third rib, presented exaggerated breath sounds and crackling râles. Below the third rib the breath sounds and voice sounds were much diminished.

The abdomen was slightly distended. There was no rigidity or tenderness except on deep palpation in upper right quadrant, where there was slight tenderness. No masses were palpable. The liver extended $2\frac{1}{2}$ cm. below the costal margin. The spleen and kidneys were not palpable. The bladder was not distended.

This patient came under my care in the receiving ward; and believing the family physician's diagnosis of empyema to be correct, and after consultation with Dr. Ashhurst over the telephone, a needle was inserted at the angle of the scapula, in the seventh right interspace, in order to relieve the urgent symptoms. After the needle had gone in about $2\frac{1}{2}$ cm. a grayish, purulent material was withdrawn, having a foul odor, not the odor of colon bacillus, however. The needle was then attached to an aspiration outfit and 580 cc. of pus was evacuated. Following the removal of pus the coughing stopped entirely, the cyanosis became much less marked, respirations much improved, the child was able to lie down (which he could not do before without a violent attack of coughing with marked cyanosis) and he was apparently much better. The child soon went to sleep and slept until about 7.30 the next morning (about eight hours), when he became restless. The pulse was very weak. The boy died at 8.30 A.M.

A necropsy was performed, for the report of which I am indebted to Dr. C. Y. White.

The pathological diagnosis was bronchopneumonia, acute pleurisy (fibrinous), subdiaphragmatic abscess, gangrenous appen-

ditis, localized suppurative peritonitis, acute diffuse nephritis and acute toxic splenitis. The appendix was about 7 cm. in length, was retrocecal and pointing toward the liver and gangrenous. From it ran a straight tract, which terminated in a large subphrenic abscess, which was intraperitoneally situated in the right posterior intraperitoneal subphrenic space. About 100 cc. of pus still remained in the abscess cavity, which by the adhesions was apparently of about two weeks' duration. The needle, which supposedly was passing through a thickened pleura, had perforated the diaphragm without entering the pleura and gone into the abscess cavity, which was bounded above by the diaphragm, below by the upper surface of the posterior portion of the right lobe of the liver, in front by the right lateral ligament and on the left by the reflection of parietal peritoneum covering the right surface of the vena cava.

In 1914 Dr. Ashhurst had admitted to his service another case of appendicitis, complicated by subphrenic abscess, which, as it has not been reported before, is included in the present report.

CASE II.—A boy (J. M.), eleven years of age, admitted November 13, 1914. The family history was negative. The patient had had an attack similar to the present one three years ago. Four days before admission the child was seized with cramplike pains in the abdomen, following an indiscretion in diet. The pain was at first generalized over the abdomen, but later became localized to the right iliac fossa. The bowels were constipated and the patient vomited after the beginning of the pain. There were no pulmonary cardiac or genitourinary symptoms.

The physical examination was negative, except the abdomen, which was somewhat distended throughout; liver and spleen not palpable. On light palpation there was some rigidity and tenderness in the right iliac fossa. By pressure there was evidence of a mass in the same locality. Peristalsis was active throughout and there was gurgling in the right iliac fossa. There were no scars or hernia. The external genitalia were negative. The temperature was 99° F.; pulse, 128; respirations, 24; urine, negative.

A diagnosis of acute appendicitis with abscess formation was made, and Dr. Ashhurst operated immediately, finding an appendiceal abscess with a gangrenous and perforated appendix. The appendix was removed and the abscess opened and drained; a rubber tube was placed to the pelvis and an iodoform drain to the stump of the appendix.

The boy was put in the Fowler position and was given continuous enteroclysis. He did quite well, having the drains removed in a few days, temperature and pulse being normal. He continued to improve until the eighth day after the operation when his temperature rose to 101° F. and his leukocytes were found to number 27,500 of which 81 per cent were polymorphonuclear. The next day his temperature was still high.

The operative incision was healed except for a superficial granulating area in its lateral half, there being no sinus and no discharge. There was no tenderness here or elsewhere, except high in the right loin, over the lower right ribs and at the costal margin in the mid-axillary line. There was slight but distinct pitting of the skin on pressure over these regions, but none elsewhere. On deep inspiration it appeared that the right costal margin moved further away from the midline than did the left (Hoover's sign). The lungs were negative.

A diagnosis of subphrenic abscess was made, and operation done the same day, November 22, 1914. A finger inserted into the incision of the first operation found no pus pockets here, but dense adhesions walling off the right flank. So a small gridiron incision was made at the edge of the ribs in the midaxillary line. When the thickened peritoneum was opened, the ascending colon presented. This was packed off and a subphrenic abscess between the liver and diaphragm was evacuated by burrowing upward with the finger. It contained about 50 cc. of creamy, inodorous pus. The culture showed short chains of streptococci. A tube was placed for drainage and the wound drained for several days. The temperature gradually subsided and the boy made a good recovery and was discharged with both wounds healed entirely, twenty-four days after the second operation. When seen nine months later he was free from symptoms.

COMMENT.¹ The statistics of Lance (1909) on subphrenic abscess comprising almost 1000 cases indicate that about 20 per cent are caused by appendicitis, 30 per cent by lesions of the stomach and duodenum, 13 per cent by lesions of the liver or gall-bladder and 37 per cent by miscellaneous affections (pancreas, spleen, large intestine, pleura and other organs).

Appendicitis may give rise to subphrenic abscess in various ways. It occurred in 20 out of one series of 2400 cases of appendicitis under the care of Dr. John B. Deaver, 4 of the patients recovering. The intraperitoneal variety was present

in two-thirds of 106 cases analyzed by Eisendrath. He found recorded only 6 left-sided cases of subphrenic abscess due to appendicitis.

According to Barnard, who fully discussed the subject in 1908, special attention should be paid to the following points in diagnosis:

1. Previous history (usual causes of the condition, *e. g.*, gastric or duodenal ulcer, appendicitis, hepatic abscess or other conditions).
2. Character of onset.
3. Constitutional signs of pus.
4. Abdominal signs and symptoms, including bulging during respiration, tenderness, rigidity, dulness or tympany due to perforation of air-containing viscus. A swelling due to subphrenic abscess is immobile because fixed by adhesions.
5. *Thoracic Signs and Symptoms.* Most important are dulness associated with upward displacement of lung, diminution or absence of breath sounds, vocal resonance and vocal fremitus. Amphoric resonance of abscess contains air. Apex-beat of heart may be displaced upward, but seldom laterally. Hoover's sign is of value in differentiating between empyema and subphrenic abscess (if the abscess is subphrenic the excursion of the costal border on the affected side is increased, being decreased on the affected side if due to empyema).
6. Fluoroscopic examination shows fixity or lessened mobility of the diaphragm on the affected side.
7. Aspiration is dangerous unless followed by immediate operation; therefore, should not be done until patient is ready for any operation that may seem proper.

In the first case reported herewith, the subphrenic abscess was in direct continuity with the gangrenous appendix; in the second case, as is more usual, the abscess appeared as a secondary complication, probably being due to direct spread of infection before or during the original operation. If the subphrenic abscess is due to spread of infection along the retroperitoneal lymphatics, it seldom gives rise to symptoms so soon after, the first operation. Two other cases of appendicitis complicated by subphrenic abscess have been reported by Dr. Ashhurst:² in the first the subphrenic abscess developed before operation, from direct intraperitoneal spread of infec-

tion; in the second it did not develop until six months after operation. In the total 4 cases, 2 patients recovered and 2 died. These were observed in a series of 200 cases of appendicitis with complications (abscess, diffuse peritonitis, gangrene, etc.), requiring drainage of the wound.

REFERENCES

1. Deaver and Ashhurst: Surgery of the Upper Abdomen.
2. Ashhurst: Transactions of Philadelphia Academy of Surgery, 1911, xiii, 154-157.

VASOMOTOR AND PILOMOTOR MANIFESTATIONS

THEIR LOCALIZING VALUE IN TUMORS AND LESIONS OF THE SPINAL CORD: A REPORT OF THIRTEEN VERIFIED CASES¹

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CLINICAL means that permit more accurate localization of lesions of the spinal cord are of extreme value. This paper calls attention to a new sign and its significance as shown in thirteen verified cases, recorded herewith. It has proved of great value in determining the exact level for operation when sensory manifestations were incomplete or misleading, and in each case the upper level of vasomotor manifestation corresponded to the upper level of the lesion disclosed by operation or the roentgen-ray.

VASOMOTOR MANIFESTATIONS. The patient should be examined after a period of rest in bed, and should be free from clothing which may produce constriction or irritation to the skin. The patient is placed in diffuse daylight of moderate intensity, the possibility of shadows being avoided, the covering is quickly removed, and the entire body scanned carefully for vasomotor manifestations. This may best be determined at a distance of from 3 to 6 feet from the patient, and careful attention should be paid to the presence of areas of hyperemia, which are suggested by a broad band of flushing about the chest or abdomen, demarcated above by the normal flush of the skin.

The direction of this band of vasomotor manifestation is in

¹ From the Department of Neurology, University of Pennsylvania, and the Institute for Investigation of Nervous and Mental Diseases, D. J. McCarthy Foundation. Reprinted from the Archives of Neurology and Psychiatry, 1928, xix, 31-46. Presented before a meeting of the Philadelphia Neurological Society, December 17, 1926, and the American Neurological Association, May 26, 1927.

striking accord with the segmental zones of Henry Head. Several variations of the phenomena may exist, and all have been found to be of equal localizing significance, indicating an abnormal instability in this area. The skin areas above and below the line of demarcation may vary in texture and in the degree of light reflection, if the patient is observed from an angle such as from the head or from the foot of the bed. Diffuse daylight should be used in all instances, as artificial light frequently gives rise to misleading effects.

The actual line of demarcation may assume a feather-edged appearance and may be no larger than the diameter of a piece of twine, extending in a serpentine fashion at right angles to the axis of the body until the axillary line is reached, and then coursing upward at an angle of about thirty degrees toward the spinous processes. The zone of demarcation in cases in which the history indicates a long-standing involvement of the cord may simulate the difference between a slight sunburn and normal skin, so that the area below the level of the lesion appears to be slightly pigmented, and this effect may extend over a large area of skin below the line of demarcation, giving the appearance that the patient had been overexposed to the weather.

The examiner will be struck by the rapid evanescence of the vasomotor reaction in the zone under observation. During the initial period of observation a flushing should appear at the level noted, similar to that seen in slight hyperemia following the application of heat to the skin. If the patient is exposed to a draught or cold, it may be noted that this area blanches rapidly, giving rise to an apparent ischemia or to a zone distinctly white in comparison with the normal areas above.

Three patients in this series were negroes, with deep pigmentation of the skin. Even in them the vasomotor level was distinctly demarcated, being especially noticeable by the "light reflex" when viewed from an angle, the area above giving the soft, oily appearance of normal skin; whereas, in the zone of vasomotor disturbance, the surface was more velvety, apparently having lost some properties of secretion.

In eleven cases of this series I was able to trace out, before making the sensory examination, the vasomotor line with a

pencil on the surface of the skin. The level of vasomotor demarcation was found to coincide exactly with the first signs of sensory change, which began with this line and became increasingly more definite below this level.

When the apparent vasomotor demarcation has been determined by the eye, confirmation of this zone can be intensified by heat. A large bath towel wrung out in hot water should be spread over the entire chest and abdomen, including the areas above and below the suspected line of demarcation. After permitting this application of moist heat to remain for about five minutes, the skin surface is again exposed, and it will be noted that a zone of hyperemia has been induced by the heat both in the normal areas and in the areas below the line of demarcation.

Careful observation may now reveal such intensification of the features of the skin areas below the level of demarcation, as seen in Fig. 5, that photography of the point of change in skin reaction is possible. The area of skin below the line of demarcation shows fluctuating vasomotor properties, hyperemia rapidly fading into ischemic zones and then reappearing in characteristic blushing. After the normal skin areas have returned to their original color, a distinct zone of hyperemia at the upper line of demarcation may persist for several hours.

After a suitable time has elapsed following the vasomotor determination, the careful testing by a blunt object to determine the upper zone of hyperalgesia gives a line that exactly corresponds with the upper level of vasomotor demarcation and may appear one or more segments above the true zone of objective sensory disturbance.

As is evident in Case IV, the vasomotor manifestations are probably radicular, and are therefore of extreme value in giving the exact level of the upper root irritated by the lesion. As pain in the root is not always a symptom in tumor of the cord, the vasomotor level indicates the site of involvement of the root. Frequently, the segmental level of involvement of the cord demonstrates itself objectively, much lower on the skin areas than the symptoms in the sensory roots, as these roots in the lumbar cord may have to pass the lesion to gain their point of exit below. This sign, therefore, becomes of

increasing value to the surgeon for properly localizing the level of operative intervention.

PILOMOTOR MANIFESTATIONS. The pilomotor reflex is obtained by flicking the skin with a cold towel, or by the use of an electric fan or a current of cold air when the skin has been moistened by a cold towel. Pilomotor manifestations may also be produced by a method recently suggested by Thomas.¹ Deep pinching of the skin and the trapezius muscle at the base of the neck will produce a homolateral reflex spreading rapidly down the same side to include the lower extremities. This is a test for function of the cord and may give the level of the lesion by failing to appear below the involved segment (Case XII).

The cold-towel method that I used often denotes the level of irritation of the root by the intensity of the reaction. It is a segmental reflex and has been noted to occur below the level of a transverse lesion.

In three cases of this series, the pilomotor manifestations appeared above the level of the lesion and ended abruptly at the line of demarcation seen on the skin, being entirely lost below this area.

REPORT OF CASES

CASE I.—*Tumor of the spinal cord at the fifth thoracic segment on the left. Endothelioma. Operation with removal.*

History. Dr. R. S. M., was admitted to the University Hospital, on the service of Dr. William G. Spiller, August 31, 1926, complaining of pain in the back and abdomen, with numbness and weakness of the left leg and loss of pain sensation in the right leg. The patient had been well until 1921, when he began to have pains around the heart and paresthesias in the precordial area on the left. In 1923, there were sharp pains in the abdomen, especially in the left epigastrium, which were thought to be due to adhesions from a former appendectomy. The pain was continuous and was increased by bending over. For a month before admission, he noted numbness on both sides of the abdomen extending into the lower extremities, more marked on the right side. Three months before admission he noted a loss of temperature sense in the right leg associated with burning pain in the outer aspect of the right foot. Constipation had been present for three months, but no loss of bladder

¹ Thomas, A.: *Rev. neurol.*, 1926, i, 767.

function. Weakness of the left leg became sufficient to require the use of a cane in walking.

Neurologic Examination. There was no loss of the sense of touch; the sense of pain was definitely impaired in the right leg below the knee with slight impairment bilaterally as high as the umbilicus. The sense of position was normal. The vibratory sense was entirely lost in the left lower extremity. The temperature sense was practically lost in the right leg, and as high as the ninth interspace on the trunk. There was great impairment on the left side of the trunk as high as the eighth interspace. There was less involvement of the left lower extremity. There was definite weakness and spasticity in the left leg.

The reflexes were normal in the upper extremities; the abdominal reflexes were lost; the cremasteric reflex was normal on the right and diminished on the left. The patellar reflexes were greatly increased on each side; the Achilles reflex was increased on the left and normal on the right. There was a positive Babinski sign on the left and a negative one on the right. There was no ankle clonus.

Laboratory Tests. Roentgenographic studies and studies of the blood and urine gave negative results. The Queckenstedt test showed a partial block.

Vasomotor Tests. The vasomotor level showed a clear-cut line of demarcation at the fifth thoracic segment bilaterally, with increased pigmentation of the skin areas below the level of the lesion. The vasomotor zone of demarcation corresponded exactly with the upper level of hyperalgesia and was in the area of the precordial pain. A definite point of tenderness was found over the third dorsal spinous process.

Diagnosis. The condition was diagnosed as tumor of the spinal cord at the fifth thoracic segment on the left.

Exploratory Laminectomy. Operation was performed by Dr. Grant, October 6, 1926, and disclosed an intradural and extradural endothelioma below the level of the fourth thoracic lamina with pressure on the cord at the level of the fifth and sixth thoracic segments, involving the root of the fifth thoracic segment on the left.

CASE II.—*Tumor of the spinal cord at the level of the fifth thoracic segment on the left. Endothelioma. Operation with removal.*

History. Mrs. W. M., aged fifty-three years, was admitted to St. Agnes Hospital, Philadelphia, on the service of Dr. D. J. McCarthy, October 2, 1926, complaining of numbness and weakness of the lower extremities. She had been well until sixteen months before, when, while standing beside the mantelpiece in her room,

she suddenly fell to the floor because of weakness of both lower extremities. There was incontinence of the bowels and the bladder. She had been confined to bed and had been unable to walk without aid or assistance since. The numbness and weakness persisted, but the bladder symptoms improved, until five weeks before admission, when she again noted an increase in the symptoms, with loss of bladder and rectal control. During the five weeks previous to admission there was rapid progression of symptoms, with gradual increase in the numbness, so that at the time of admission it extended as high as the waist. The weakness in the lower extremities became marked and she was unable even to move the limbs while in bed. Three months before admission she noted a sharp and shooting pain over the left side of the chest in the region of the precordium.

Neurologic Examination. There was no definite weakness of the muscles of the lower portion of the trunk. The lower extremities showed almost complete paralysis. Spontaneous reactions of defense were present.

The reflexes in the upper extremities were active and equal. The abdominal reflexes were lost bilaterally. The patellar reflex was greatly exaggerated on the right and increased on the left. The Achilles reflex was slightly increased on both sides. There was bilateral abortive clonus, with bilateral Babinski and Oppenheim signs.

The sense of position was lost in both lower extremities. The sense of vibration was lost below the thorax. The temperature sense was lost below the ninth thoracic segment. Tactile sense was lost below the fifth thoracic segment on each side. Pain sense was lost below the fifth segment on the left and the seventh on the right (Fig. 2).

Vasomotor and Pilomotor Manifestations. A zone of vasomotor reaction was present over the chest and posteriorly at the lower angle of the scapulas. There was definite hyperemia in the lower two-thirds of the breast on the left side. Posteriorly, the vasomotor line of demarcation seemed to be slightly lower on the right than on the left. The zone of hyperalgesia corresponded exactly with the vasomotor manifestations. There was distinct tenderness to pressure over the third dorsal spinous process (Figs. 1 and 2).

The Queckenstedt test showed a complete block.

Exploratory Laminectomy. Operation was performed by Dr. Fay, November 9, 1926, and disclosed an endothelioma on the left side of the cord, at the level of the fifth thoracic segment, which was removed (Fig. 3).

Comment. The vasomotor level in this case was of distinct localizing value and accurately outlined the upper level of the lesion, as shown by operation.

Case III.—*Tumor of the spinal cord, second to sixth segments—subdural lipoma over the dorsal surface of the cord. Operation with partial removal and recovery.*

History. Miss M. G., aged twenty-one years, was admitted to the Episcopal Hospital, Philadelphia, on the service of Dr. George Wilson, October 25, 1926, complaining of pain over the heart, with numbness and weakness of the lower extremities. She had been well until six weeks before, when she noted pain around the region of the heart and over the left side of the chest. She was confined to bed for one week, but noted on sitting up that pain was present, especially when she bent forward. Five weeks before admission she returned to work and noted weakness in the lower extremities when attempting to board a trolley car. The weakness progressed so that within four days she was unable to walk.

Neurologic Examination. Vibration sense was lost in the right lower extremity and as high as the costal margin. It was impaired on the left to an equal level. Position sense was lost in both lower extremities. Pain sense was impaired in both lower extremities and as high as the eleventh thoracic segment, more marked on the left; slight impairment of pain sense was noted as high as the sixth thoracic segment bilaterally. Temperature sense was greatly impaired in the right lower extremity and as high as the twelfth thoracic segment on the right. It was bilaterally impaired as high as the sixth thoracic segment. There was a definite point of tenderness over the fourth dorsal spinous process. A definite level of hyperalgesia was obtained at the fifth thoracic segment, corresponding to the zone of root pain (Fig. 6). Pain in the precordial area was accentuated by coughing and sneezing.

A Queckenstedt test showed definite evidence of spinal block.

There was marked paralysis of both lower extremities with reactions of defense.

Bilateral clonus and Babinski sign were present. The abdominal reflexes were greatly diminished, and there was an increase in all the deep tendon reflexes of each lower extremity.

Vasomotor and Pilomotor Manifestations. A definite zone of vasomotor reaction was noted on the left side, extending from the third thoracic segment to about the level of the tenth rib. Intensification of this zone by heat produced a unilateral flushing, as seen in Fig. 5, confined to the left side and extending from the upper

level of the scapulas to fade away in the lower portion of the chest. This wide zone of vasomotor reaction was difficult to interpret, but operation revealed ample cause for its wide distribution, though the unilaterality of the sign is still a matter of conjecture (Fig. 4).

Exploratory Laminectomy. The operation, performed by Dr. Fay, extended from the second to the sixth dorsal segment of the cord. A subdural tumor was disclosed, extending over the dorsal aspect of the cord throughout the area of exploration (Fig. 7). An attempt to remove the tumor failed to disclose any line of demarcation between the tumor and the substance of the cord. A portion of the tumor was taken for microscopic study and proved to be lipoma, similar in character to that reported recently by Stookey.²

CASE IV.—*Tumor of the spinal cord of the third and fourth thoracic segments, intramedullary, probably glioma. Operation with decompression.*

History. W. L., a man, aged fifty-four years, colored, was admitted to the University Hospital on the service of Dr. William G. Spiller, August 30, 1926, complaining of pain in the region of the right shoulder, numbness in the right lower extremity, and weakness of the right leg. He had been well and occupied with farming until May, 1926, when he first noted pain below the right shoulder blade, which was aching in character and gradually extended around to the anterior surface of the chest on the right side. Shortly after the onset of pain, the right leg began to feel numb and stiff. Two months before admission, weakness occurred in the right leg, so that on admission the man required a cane for walking. Constipation began one month before admission and was associated with difficulty in urination. At the time of admission, the patient complained of a girdle sensation and peculiar drawing feelings in the left leg.

Neurologic Examination. Pain sense was lost on the right as high as Poupart's ligament; above this was a zone of hyperalgesia; in the lower abdomen, from the costal margin to a point just above the nipple on the chest, pain sense was greatly diminished; pain sense was lost on the left side, including the lower extremities and abdomen, and over the thorax to a level just below the nipple. Vibration sense was impaired in the right lower extremity and as high as the thorax; it was normal on the left. Sense of position was normal in both lower extremities. Touch sense was impaired up to the level of the nipple line, bilaterally. Temperature sense corresponded

² Stookey, Byron: Intradural Spinal Lipoma, Arch. Neurol. and Psychiat. 1927, xviii, 16.

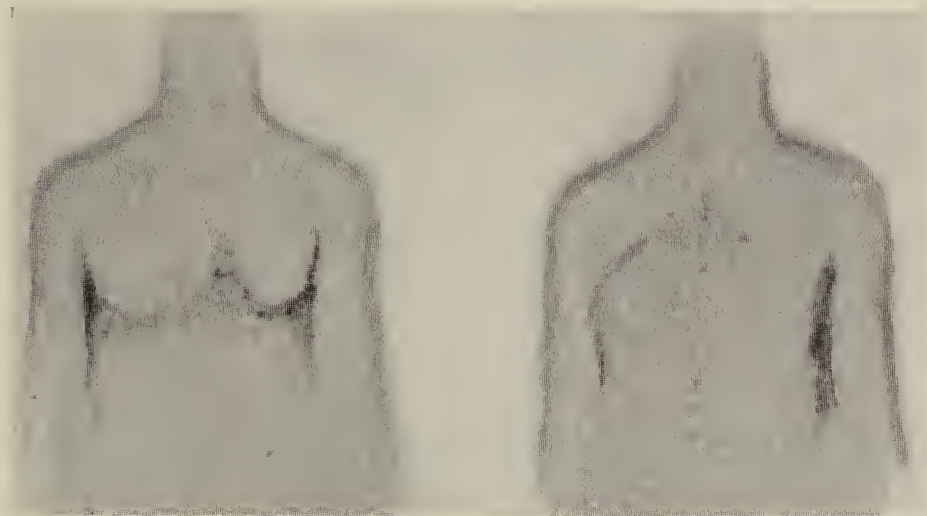


FIG. 1.—Case II. The vasomotor level as seen after the application of heat to the skin. The tumor, as seen in Fig. 3, involved the fourth and fifth thoracic roots on the left.

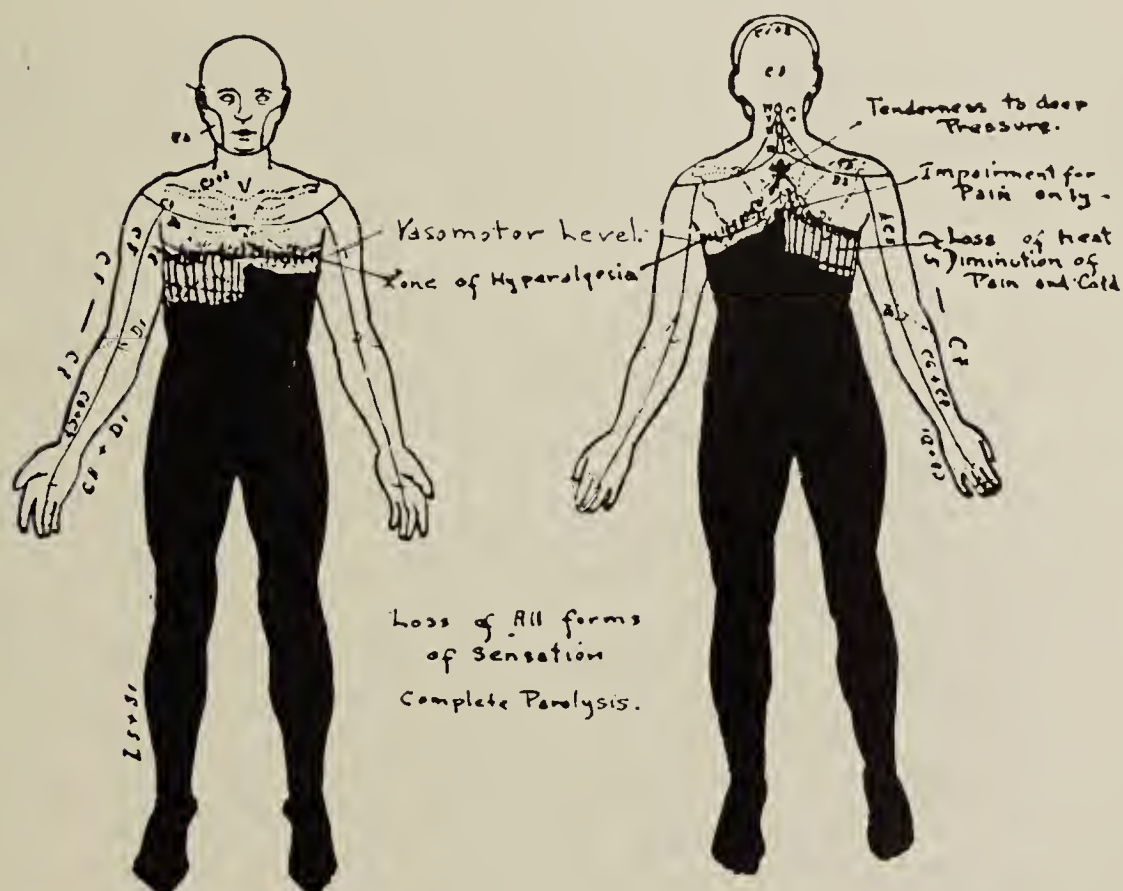


FIG. 2.—Case II. In this case the sudden onset of symptoms and the absence of definite root pains with profound sensory disturbance led to the diagnosis of myelitis. The distinct level of vasomotor manifestations, the corresponding level of hyperalgesia and the positive Queckenstedt sign led to an exploration. The tumor was found and removed from below the point of deep tenderness to pressure shown on the back.



FIG. 3.—Case II. Endothelioma of the cord involving the fourth and fifth thoracic roots on the right and the fifth thoracic segment of the cord. The tumor was completely removed at operation. The vasomotor level definitely established the upper level of the lesion.



FIG. 4.—Case III. The vasomotor response to heat shown on the left was almost entirely unilateral. The pilomotor response showed an even higher level and did not extend below the vasomotor line of demarcation on the left. The tumor, as seen in Fig. 7, was a large subdural lipoma of the cord covering the dorsal aspect with slightly more marked involvement of the left side. The wide zone of vasomotor response and the high level of the pilomotor disturbance corresponded closely to the large number of segments of the cord involved.



FIG. 5.—Case III. The wide zone of vasomotor response after the application of a hot towel to the back. In the lower illustration the zone has been marked out to make apparent what is difficult to portray photographically. The zone includes the third thoracic segment extending onto the arm and into the axilla, also down the back to the sixth segment. In this case the response was entirely confined to the left side. Identical photographs.

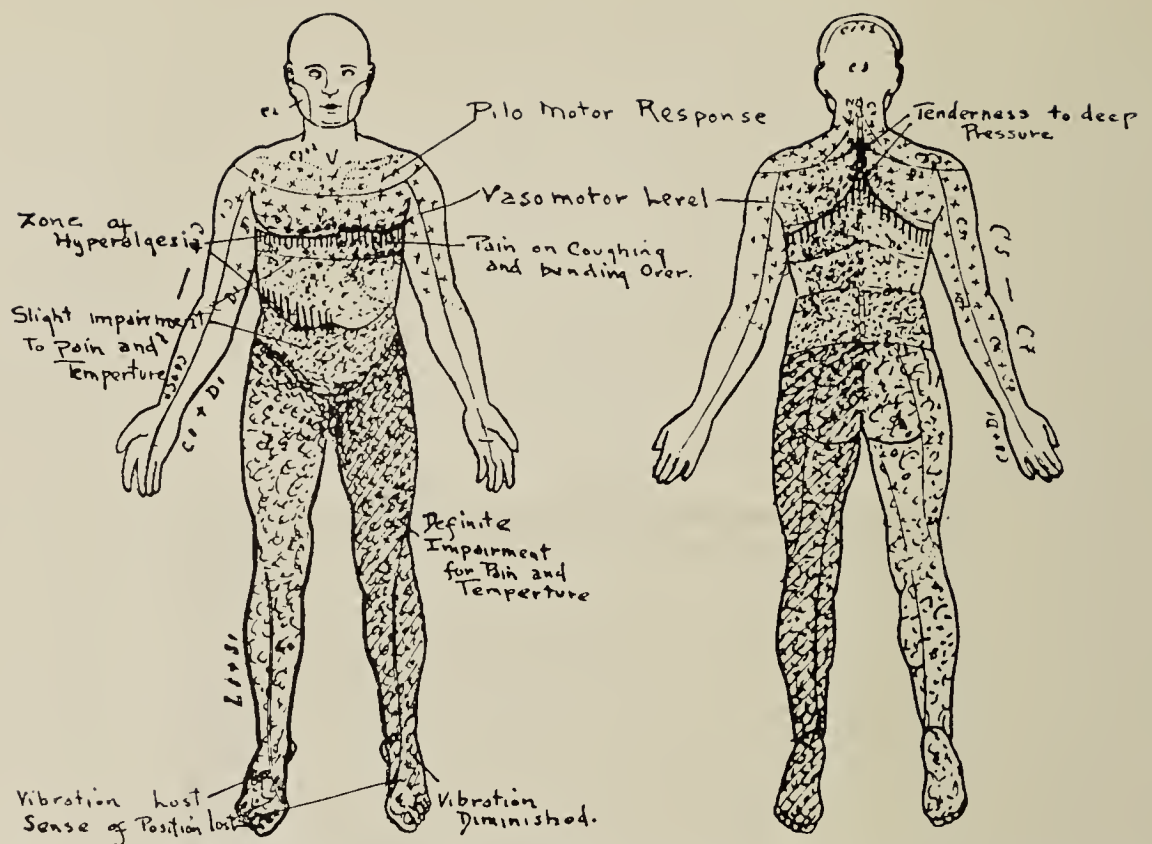


FIG. 6.—Case III. The sudden onset of symptoms, the early sensory changes and the wide zone of vasomotor change led to some uncertainty as to the nature and extent of the lesion. The positive Queckenstedt test and the area of deep tenderness to pressure on the back were of assistance in diagnosing the lesion. The abrupt level in which the pilomotor responses ceased coincided with the upper level of the vasomotor change and the zone of hyperalgesia and thus determined the level of operative exposure.



FIG. 7.—Case III. Appearance of subdural tumor, extending over the dorsal aspect of the cord throughout the area of exploration.

to the levels of pain sense but was apparently more greatly disturbed in the left lower extremity than in the right. There was an area of tenderness to deep pressure over the fourth and fifth spinous processes.

There was definite weakness of the right lower extremity.

The abdominal reflexes were lost; the patellar reflexes were greatly exaggerated, especially on the right; the right Achilles reflex was exaggerated, the left normal; there was no Babinski sign and no clonus.

Laboratory Examination. The laboratory and roentgen-ray reports were essentially negative. A Queckenstedt test was positive for a partial block.

Vasomotor and Pilomotor Manifestations. A definite zone of vasomotor demarcation was noted in this case even in the presence of the deep pigmentation of the skin characteristic of the patient's race. The skin appeared oily and normal above the zone of demarcation, but there was a distinct tendency toward a velvety and dull luster to the skin below. The zone of demarcation on the right appeared at a level just above the nipple; on the left it was just below the nipple. The addition of heat to accentuate the vasomotor zone made the line of demarcation even more evident. The application of cold to the skin produced a pilomotor reflex that was intense in the area above the zone of vasomotor demarcation; it ceased abruptly at this line and did not extend below into the level of sensory disturbance.

Diagnosis. The condition was diagnosed as tumor of the spinal cord on the right at the level of the fourth and fifth thoracic segments.

Exploratory Laminectomy. The laminæ were removed by Dr. Frazier, December 18, 1926, over the second, third, fourth and fifth thoracic segments. Marked enlargement of the cord was disclosed in segments three and four; the process was cystic and probably gliomatous. A decompression was made and recovery followed.

Comment. At the operation it was necessary to section the third and fourth thoracic roots in order to explore the anterior surface of the cord. Following this operative procedure, the patient presented a new level of vasomotor manifestations; the zone of vasomotor demarcation had risen to include the third thoracic segment, extending out on the arm sufficiently to include the axillary region and over the upper border of the chest. The old line of vasomotor demarcation was still evident by slight increase of pigmentation of the skin, but this was not so marked as formerly.

It would appear therefore, that the vasomotor reaction noted on the skin in this case was probably a radicular pressure phenomenon with loss of vasomotor control to the dermatome involved.

CASE V.—Intramedullary tumor of the cord at the level of the fifth, sixth and seventh thoracic segments exploratory laminectomy with decompression and incision of the tumor mass—recovery.

History. Miss E. K., aged seventeen years, was admitted to the Orthopedic Hospital, Philadelphia, on the service of Dr. T. H. Weisenburg, September 26, 1925, complaining of weakness and numbness of the lower extremities for the past six months. She had been apparently well until five years before. At that time the mother noted staggering in gait. The patient frequently fell or stumbled when going upstairs. Examination by a physician disclosed a curvature of the spine, and a cast was applied for three months. Symptoms entirely disappeared for four years. One year before admission she again noted staggering, with coldness and numbness in the feet, which gradually extended upward until at the time of admission the area was as high as the waist. The weakness of the lower extremities had also increased. There was a girdle sensation in the lower portion of the chest. Bladder symptoms began four years before, with frequency and urgency, and persisted throughout the illness. There was no incontinence.

Neurologic Examination. There was bilateral loss of the abdominal reflexes. Marked spasticity of the lower extremities was present, more pronounced on the left. The patellar and Achilles reflexes were exaggerated bilaterally. Ankle clonus was present on both sides, more marked on the right. Bilateral Babinski and Oppenheim signs were elicited. The sense of vibration was lost in both lower extremities. Sense of position was lost in both lower extremities. Temperature sense was lost on the right as high as the fourth intercostal space and greatly diminished on the left as high as the fifth intercostal space. Tactile sensation showed marked diminution in the lower extremities as high as the costal margin. Temperature sense was involved in a similar degree and area as the pain sense. A level of hyperalgesia corresponded to the upper level of vasomotor manifestation. A Queckenstedt test was positive.

Vasomotor Manifestations. A vasomotor level was determined on the left side over the chest in about the third and fourth interspace.

Diagnosis. The condition was diagnosed as tumor of the cord at the level of the fifth and sixth thoracic segments, at the level of the fourth root.

Exploratory Laminectomy. Operation was performed by Dr. A. P. C. Ashhurst, December 2, 1925, and disclosed an intramedullary tumor of the cord extending from the fifth to the seventh thoracic segments. Decompression was made, with closure, and recovery followed. A second operation was performed by Dr. A. P. C. Ashhurst and Dr. Fay, February 4, 1926, with exposure of the tumor and the normal area of the cord above. Cordotomy was performed, with section of the anterolateral columns above the tumor mass for relief of pain. Incision into the tumor revealed a gelatinous center which was thought to be glioma. Further decompression was secured with recovery.

Comment. The tumor was found to be situated in the region from the fifth to the seventh thoracic segments at the first operation; it had extended slightly higher at the second operation and involved the fourth thoracic segment.

The vasomotor sign still persists.

CASE VI.—*Gunshot wound of the spinal cord, with destruction of the sacral portion; the bullet entered the midline at the level of the first lumbar spine; the roentgen-ray disclosed it lodged at the level of the ninth dorsal vertebra.*

History. Mr. W. D., aged twenty-four years, was admitted to the Philadelphia General Hospital on the service of Dr. George Wilson, December 1, 1926, complaining of paralysis of both lower extremities following a gunshot wound of the spine. He had been well until October 16, 1926, when he was shot by a policeman and was removed to the hospital in an unconscious condition. The bullet had entered the middle of the back at the level of the first lumbar spine, taking an upward course. The roentgen-ray revealed the bullet lodged at the level of the ninth dorsal vertebra in the region of the liver. The patient had complete flaccid paralysis of the lower extremities, with marked wasting and atrophy of all muscle groups of the legs and around the buttocks. There was complete anesthesia below the second lumbar segment, including the sacral area, with incontinence of bowels and bladder.

Neurologic Examination. Symptoms of a transverse lesion of the cord at the second lumbar segment, with destruction of the sacral and lumbar portion below this level, were disclosed. There was complete anesthesia and paralysis with loss of patellar and Achilles reflexes and marked atrophy. From the level of the eighth thoracic dermatome on the lower portion of the chest to Poupart's ligament there was hyperalgesia, associated with spontaneous root pains in this area. Below Poupart's ligament, beginning at about

the first lumbar segment, there was marked impairment to all forms of sensation on each side. There was loss of bladder function with anesthesia of the scrotum and penis, and incontinence of feces.

Diagnosis. The condition was diagnosed as destruction of the sacral and lower lumbar cord, by gunshot wound, causing traumatic myelitis involving the roots as high as the eighth dorsal.

Vasomotor and Pilomotor Manifestations. There was a definite line over the anterior surface of the body and also toward the posterior lateral aspect of the abdomen, where a slight change in hyperemia of the skin was evident. The upper level of the line reached a point just under the costal margin and in about the eighth dorsal cutaneous segment. Vasomotor and pilomotor reflexes in this region could be produced by irritation to the skin, which gave an evanescent fluctuation of hyperemia and ischemia. At the upper line of vasomotor demarcation the objective sensory changes became evident. Above this line sensation was normal, but when a blunt object was drawn over the surface of the skin hyperalgesia was immediately encountered at the level of the vasomotor disturbance. Hyperalgesia was present over the abdomen to the level of the first lumbar segment.

Application of heat to the abdomen and chest produced marked hyperemia in the zone of hyperalgesia; that is from the eighth thoracic to the first lumbar segment. In this zone, there was marked flushing following the application of the hot towel with a definite upper line of demarcation at the eighth dorsal segment. Cold applied to the skin produced a marked pilomotor reflex in the zone of hyperalgesia, as well as above, so that the only demarcation between the pilomotor reflex above and below the level of the lesion was manifest by a more intense reaction of the skin below the eighth dorsal segment and extending down to the first lumbar segment (hyperirritability). Above the eighth dorsal segment, in the normal response of the skin, the pilomotor influences were distinct but not as marked as in the zone of hyperalgesia.

There was an intense and marked reaction to cold which produced "goose flesh" that was distinct from that produced in the normal areas above. The application of cold in the form of ice produced a white line of ischemia along the upper level of demarcation. This line was so distinct and definite that it could be marked out on the abdomen and closely followed the upper line of vasomotor evanescence. The vasomotor demarcation exactly coincided with the upper level of sensory changes. Furthermore, the line as accentuated by cold was identical with the line of demarcation as

noted on the patient's abdomen before the application of either heat or cold and detectable at a short distance from the patient by the eye alone.

Comment. In this case the upper level of sensory disturbance was anticipated and a line was drawn on the chest before the sensory examination was made. It was found to coincide exactly with the upper level of hyperalgesia, and, on roentgen-ray studies, the bullet was shown to be opposite the ninth thoracic vertebra so that involvement of the eighth thoracic root and those below seemed evident.

CASE VII.—*Tumor of the spinal cord at the first lumbar segment on the right extending 7 cm. upward to involve the ninth thoracic root. Sarcoma. Operation with removal.*

History. F. W. B., aged sixty-five years, was admitted to the service of Dr. William G. Spiller, December 27, 1926, at the University Hospital, complaining of difficulty in walking and pain in the back. The patient showed a spastic paraplegia, with bilateral clonus and Babinski sign. The sensory examination showed impairment of pain and temperature only in the lower extremities, with loss of vibratory sense to the costal margin. There was a wide zone of hyperalgesia extending from the costal margin to the inguinal region on each side.

The roentgen-ray examination of the spine showed well-marked hypertrophic spondylitis of the lumbar vertebræ. The Queckenstedt test showed a complete spinal block. The spinal fluid was xanthochromic. Other laboratory examinations gave negative results.

Vasomotor Manifestations. There was a definite vasomotor level at the eighth thoracic dermatome over the upper part of the abdomen on both sides. There was a lower level on the right side in this case that was clear cut in the first lumbar distribution on the right. This area was pigmented and showed marked hyperalgesia. It was the area in which the patient referred much of his pain.

Diagnosis. Although the sensory manifestations were not convincing and as they failed to show a clear-cut level and as hypertrophic spondylitis was thought to explain many of the symptoms, the reliability of the vasomotor sign in other cases led to the diagnosis of a tumor of the cord on the right side involving the first lumbar segment and several roots above. It was thought that the lesion might be large because of the wide zone of vasomotor manifestations similar to that seen in Case III.

Exploratory Laminectomy. Operation was performed by Dr. Grant, February 9, 1927, and disclosed a large vascular tumor on the right side of the cord extending over several segments and involving the ninth thoracic to the first lumbar roots. The tumor was removed.

Comment. The definite vasomotor manifestations in this case led to the correct localization and the decision for exploration. The intense vasomotor reaction on the right side at the first lumbar segment proved to be the point of greatest involvement of the cord.

CASE VIII.—*Tumor of the spinal cord involving the eighth, ninth and tenth thoracic roots on the left. Melanotic sarcoma. Operation with partial removal.*

History. Mrs. M. H., aged forty-five years, was admitted to the University Hospital on the service of Dr. William G. Spiller, March 28, 1927, complaining of paralysis of the lower extremities and loss of sensation. Six months prior to admission, the patient had noted weakness in both legs, which had gradually increased until two months later she could no longer walk. Numbness in the legs was noted at this time. On admission, she showed a complete transverse lesion with sensory and motor loss as high as the ninth thoracic segment.

The Queckenstedt test showed complete block. Roentgenograms and other laboratory examinations gave negative results.

Vasomotor Manifestations. There was a definite vasomotor level at the eighth thoracic dermatome, slightly higher on the left. Below this was a band of pigmentation extending to the tenth on the right and to the eleventh on the left.

Diagnosis. The condition was diagnosed as tumor of the spinal cord at the ninth thoracic segment.

Exploratory Laminectomy. Operation was performed by Dr. Grant, April 26, 1927, disclosing a melanotic sarcoma at the tenth thoracic segment on the left, involving the eighth and ninth roots. There were many small dark patches scattered over the arachnoid. The bulk of the tumor was removed.

CASE IX.—*Tuberculous spinal caries involving the sixth thoracic vertebra with transverse myelitis. Albee bone graft with partial recovery.*

History. Mrs. S. H., aged fifty-one years, was admitted to the University Hospital on the service of Dr. William G. Spiller, December 25, 1926, complaining of paralysis of the lower extremities and incontinence. Four months prior to admission, she had noted weakness in both legs. This rapidly increased and was associated with numbness, which extended as high as the costal margin.

At the time of admission to the hospital, there was great weakness of the lower extremities, with exaggeration of the deep reflexes, clonus and Babinski sign. Sensory tests showed an almost complete transverse myelitis to the level of the seventh thoracic dermatome. A zone of hyperalgesia extended to the sixth on the left and the fifth on the right.

Vasomotor Manifestations. On initial inspection at the time of admission, a definite vasomotor level at the seventh thoracic dermatome was noted, with an area of pigmentation below.

Palpation of the back disclosed a definite prominence at the sixth thoracic vertebra, with angulation and a point of tenderness to deep pressure just above.

Roentgenograms showed a destruction and a crushing of the body of the sixth thoracic vertebra.

Diagnosis. The condition was diagnosed as Pott's disease of the sixth thoracic vertebra.

Operation. A correction of the angulation and an Albee bone graft was undertaken, February 24, 1927, by Dr. Gill. The patient later regained partial function of the lower extremities.

Comment. The patient was referred with the diagnosis of tumor of the spinal cord; the slight angulation of the spine had not been noted. The vasomotor sign indicated the level of the lesion on initial inspection, and examination at this level of the back revealed the evident spinal deformity. Roentgen-ray studies made the following day disclosed the suspected vertebral disease.

In this case, the patient was a negro, and the level was seen without the aid of heat by standing at the head of the bed; the "light reflex" gave a soft, deep, velvety appearance below the level of the lesion as compared with the shining oily normal areas above.

CASE X.—*Tumor of the spinal cord, involving the first lumbar root on the right with pressure on the sacral cord. Extradural sarcoma. Operation with partial removal.*

History. H. L., aged forty-six years, was admitted to the Orthopedic Hospital, on the service of Dr. Astley P. C. Ashhurst, January 25, 1927, complaining of severe pains in the legs and in the right groin. Incontinence had developed, and there was increasing weakness in the lower extremities. Morphine was required for alleviation of the pain.

Sensory examination did not show a clear-cut level. There was loss of sensation to pain in the sacral distribution but no disturbance of tactile, position or vibration sense. Slight hyperalgesia was noted over the lumbar distribution.

The reflexes in the lower extremity were absent on each side, and atrophy of the muscles of the legs and buttocks was present.

Vasomotor Manifestations. In this case the vasomotor sign was striking and was confined to the first lumbar dermatome on the right side only. This area was distinctly flushed, and the similarity to the condition in Case VII was so marked that a lesion of the first lumbar root was suspected; this, together with the sacral disturbance placed the lesion at the level of the first lumbar vertebra, catching the root of exit on the right. It was suspected that the lesion might be extradural, and this diagnosis was made at the time.

The Queckenstedt test was positive for a complete block, and the spinal fluid showed slight xanthochromia.

Diagnosis. The condition was diagnosed as extradural tumor involving the first lumbar root and sacral cord.

Exploratory Laminectomy. Operation was performed by Dr. Ashhurst and Dr. Fay, April 28, 1927, and disclosed a vascular tumor in the region of the first lumbar vertebra invading the spinal canal and the muscle bed without. The lamina of the first lumbar vertebra was found to be soft and infiltrated. The tumor lay on the right side of the cord, and a large portion produced pressure on the dura from without.

Comment. In this case the vasomotor sign was of great value in giving the location of the tumor as well as its probable extradural position. Operation disclosed that the first lumbar root was involved in the tumor mass as it emerged from the dura. It is apparent that the interpretation of this sign of involvement of the root gave the level for operation in the lumbar region, whereas if the condition had been considered as a manifestation of involvement of the segment, a higher exploration would have been necessary.

CASE XI.—*Fracture dislocation of the first lumbar vertebra with destruction of the sacral and lower lumbar cord—twenty-eight years' duration.*

History. Mrs. B. B., aged forty-five years, was admitted to St. Agnes Hospital, on the service of Dr. Leonard Averett, March 17, 1927, complaining of trophic ulceration of the right heel, total prolapse of the uterus and transverse myelitis of the lower lumbar cord. At the age of seventeen years, the patient fell 30 feet from a balcony, sustaining a fracture dislocation of the first lumbar vertebra and a fracture of both femurs. For the past twenty-eight years she had walked on crutches, though there was complete paralysis of both lower extremities below the knees.

I saw the patient in consultation as to the advisability of hysterectomy in the presence of the neurologic manifestations.

The sensory examination showed a complete loss of all forms of sensation below the second lumbar distribution. There was complete paralysis of the lower extremities below the knees, with atrophy and loss of reflexes. Hyperalgesia was present in the area between the eleventh thoracic and the second lumbar dermatomere.

Vasomotor and Pilomotor Manifestations. There was a striking yellowish pigmentation of the skin seen in the area between the eleventh thoracic and the second lumbar dermatomeres, inclusive. This gave the appearance of an apron, and close inspection showed that the skin was more deeply pigmented. This region corresponded to the zone of hyperalgesia.

The pilomotor reflex was spontaneous in this zone on algophoric stimulation, and was increased when pinched over the base of the neck (Thomas method). These manifestations were considered to be due to irritation of the roots from a long-standing traumatic pachymeningitis.

Roentgen-ray Examination. An old healed fracture dislocation of the first lumbar vertebra was shown by the roentgen-ray.

Comment. The patient was of extreme interest in that she was seen twenty-eight years after the initial lesion. The vasomotor sign was characterized by pigmentation of the skin, which was easily apparent. This conforms to the other cases in which the condition had been present for a period of months. The flushing noted in an early lesion (fourteen hours, in Case XIII) is replaced by pigmentation in the area later on.

CASE XII.—*Angioma of the spinal cord at the seventh, eighth and ninth thoracic segments. Exploration and decompression.*

History. R. E., a boy, aged nineteen years, was admitted to the University Hospital on the service of Dr. William G. Spiller, April 20, 1927, complaining of a heavy feeling in the legs, weakness and inability to walk without crutches. One year before admission, he noted difficulty in walking and a tendency to drag the feet. Six months later, it was necessary for him to use crutches, and shortly after this he noted incontinence of urine.

Sensory examination showed hyperalgesia at the seventh thoracic skin segment, extending down to the ninth. Pain and temperature sense were greatly impaired on the right side below the ninth thoracic segment. There was impairment of vibration, position and touch sense in both lower extremities and as high as the umbilicus. The reflexes were exaggerated, with bilateral clonus and Babinski sign.

Vasomotor and Pilomotor Manifestations. The vasomotor level could be seen at the eighth thoracic dermatomere extending around

the body. Some slight flushing at times occurred as high as the seventh dermatome. There was a band of pigmentation below this level in the ninth to the eleventh thoracic skin segments.

The pilomotor reflexes were of great interest. When the skin at the base of the neck was pinched in the region of the trapezius muscle, a homolateral pilomotor response was brought out at once on the right side and extended to the lower extremity. When this test was applied to the left side, the pilomotor manifestation did not reach below the eleventh thoracic dermatome. This was evidence that stimulation from the upper cord was interrupted on the left side at about the eleventh thoracic segment.

Diagnosis. The condition was diagnosed as probably an intramedullary lesion of the spinal cord at the ninth thoracic segment.

Exploratory Laminectomy. Operation was performed by Dr. Frazier on June 3, 1927. An extensive angioma of the cord was found, involving the sixth, seventh, eighth, ninth and probably the lower segments of the cord. Decompression without removal was performed.

Comment. The pilomotor manifestations in this case gave a clue to the intramedullary possibilities of the lesion and, together with the disturbance of pain and temperature sense on the right, seemed to indicate greater involvement of the cord on the left. I was unable to determine if the operative manifestations would bear this out.

CASE XIII.—*Fracture dislocation of the eleventh thoracic vertebra—transverse myelitis and acute meningomyelitis. Exploration and decompression.*

History. N. K., a man, aged twenty-one years, was admitted to my service at the Polyclinic Hospital, January 1, 1926, with a broken back and complete paralysis of the lower extremities. The patient was found unconscious and paralyzed after receiving an injury in an unknown manner about fourteen hours previously.

Examination showed a complete motor and sensory paralysis below the waist, with a zone of hyperalgesia between the eighth and tenth thoracic dermatomes.

Vasomotor Manifestations. A definite band of flushing at the level of the umbilicus extended around the body and was fluctuating and feather-edged. Hyperalgesia was most intense at this level.

Roentgen-ray examination showed a fracture dislocation of the eleventh thoracic vertebra, with fracture of the lamina. The Queckenstedt test was positive.

Exploratory Laminectomy. Operation was performed at once by Dr. Fay, and the lamina of the eleventh thoracic vertebra was

found fractured and depressed, pressing deeply on the cord. After removal of the fragments, the cord was exposed and a localized intense swelling was disclosed. Decompression was effected.

COMMENT. In the 13 cases reported in this paper, the vasomotor level was found to correspond exactly with the upper level of sensory disturbance and to indicate the highest point and extent of the lesion in the spinal cord. It has therefore proved of great value in accurately determining the level for operative intervention. It was found to be more reliable than the sensory level as determined in Cases I, III, VII and X, and in all the cases studied it proved to be the most accurate sign for determination of the level. Four other important factors have been present in each case: (1) a definite history of involvement of the cord suggesting a localized lesion; (2) either a complete or partial Queckenstedt test was obtained in each case (except in Case VI, which was caused by a gunshot wound the extent of which was evident from the roentgen-ray examination and in Cases IX and XI, in which roentgenograms disclosed the lesion); (3) careful testing for a zone of hyperalgesia at the upper level has shown it corresponded to the level of vasomotor change; (4) tenderness on deep pressure over the spinous processes was found near or just above the level of demarcation, as noted by the vasomotor change and hyperalgesia.

The reliability of the sign has been so definite that it was the determining factor in deciding on operative intervention in Cases II, III, VII and X in which the history of onset was so abrupt as to lead to the belief that tumor of the cord was unlikely. Since February, 1926, when the significance of this sign became apparent, 23 verified cases have been observed, 13 of which are here reported. In all, the vasomotor level indicated the upper level of sensory demarcation and the highest point of involvement of the cord in cases of discrete lesions, such as tumor of the cord, or in traumatic or vertebral disease. It has become possible with the four contributing factors noted to determine accurately the sensory level of disturbance of the cord by inspection of the skin on the patient before sensory determinations have been made. As yet, the

observations have been confined to manifestations shown by lesions of the thoracic and lumbar cord.

The patients have been observed carefully for skin manifestations, such as sweating and oiliness of the skin, hyperemia, ischemia and "goose flesh," with suitable tests to determine the value of each. It has been found that the vasomotor sign is most reliable and has been constant in all cases. The pilomotor manifestations are next in value but have not been found confined distinctly to the level of vasomotor or sensory disturbances in all cases.

At times two levels have been observed, the upper level determining the point of highest irritation of the root, with slight vasomotor changes in the skin below the point of involvement. The lower level has been characterized by pigmentation, which is definite and corresponds with the level of beginning objective sensory disturbance in cases in which the lesion has persisted for more than three months. The vasomotor levels may be close or several segments apart.

In attempting to determine an explanation for the manifestations noted regarding the pilomotor and vasomotor reflexes, the most comprehensive study dealing with this work was found in the recent monograph by Thomas, whose work on the sympathetic system satisfactorily explains the mechanism of the vasomotor and pilomotor disturbances in these cases.

Horsley utilized the vasomotor properties of sweating in his patients as a means of localization of a lesion of the cord. Thomas and his followers pointed out the values of vasomotor and pilomotor responses in vertebral and extravertebral lesions involving the sympathetic fibers. Foerster used the vasomotor response to stimulation of a dorsal root as a means of mapping out the segments supplied to the skin surface and thus determined the area of the dermatome under consideration. The presence of this vasomotor sign in tumors or lesions of the spinal cord has been of distinct value in this series, demonstrating the highest point of involvement of the root by the lesion and thus indicating the exact level for surgical intervention.

CONCLUSIONS. 1. A simple method for the accurate determination of the upper level of lesions and tumors of the spinal cord is outlined.

2. In 13 verified cases which are reported the vasomotor line of demarcation has proved to be the most reliable sign; in each case it has determined the exact level of localization.

3. In all the cases a history of a lesion of the spinal cord was evident. The Queckenstedt test was positive for partial or complete block. The zone of hyperalgesia corresponded exactly with the upper level of vasomotor demarcation. A point of tenderness to deep pressure was found in each case over the spinous processes at the level of the lesion.

4. From the surgical standpoint, the vasomotor level indicates the root level for operative exploration and, in those cases coming to operation, has proved to be correct, even though the sensory tests in some would indicate a lower position of the lesion in the cord.

5. The vasomotor line of demarcation will frequently indicate the level of the lesion before the development of sensory or motor symptoms of focal value.

6. This vasomotor sign, when associated with a positive Queckenstedt test and a zone of hyperalgesia corresponding to the level of vasomotor demarcation (trauma and evidence of vertebral disease having been ruled out by history or roentgen-ray) has been found so far only in lesions producing pressure on the spinal cord.

7. The earliest appearance of this sign was noted fourteen hours after injury to the cord (Case XIII) and the longest duration was twenty-eight years following a spinal lesion (Case XI).

CLINICAL STUDY OF VASCULAR SPASM*

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THAT the condition of vascular spasm is a rare one is well attested by a careful review of the literature; in fact, its rarity is remarked upon by no less a person than Sir Clifford Allbutt. It is mentioned especially in conjunction with intermittent claudication, which is in reality one local example of the condition. As accuracy in diagnosis is the aim of all of us, as upon it depends both treatment and the latter's results, vascular spasm, or angiospasm, must be borne in mind when one is examining cases in which there is present either a hemiplegia or a monoplegia or aphasia—not to mention edema of an extremity, acute phlebitis, cellulitis or lymphangitis.

With these facts in mind, it seemed to me worth while to record a short series of cases which have come under my observation within the past two years.

Angiospasm is seen most frequently in arteriosclerotic patients, though it is not confined entirely to them. Osler is of the opinion that Peabody best explained the condition, when discussing the cerebral symptoms of arteriosclerosis, by stating that there occurs spasmodic contraction of the muscular coats of the middle cerebral artery, or its several branches, which in connection with the narrowed lumen (caused by the sclerosis) leads to a decreased blood supply to the brain tissue supplied by the affected artery. The condition may be of short duration and frequently recurs. According to Allbutt, it may terminate in occlusion and thrombosis, generally partial, owing to the lateral projection of the proliferated coat. That Peabody's explanation, noted above, is a correct one, will be brought out in an article soon to appear; a surgeon exposed a vessel in spasm.

* Reprinted from the Medical Journal and Record, 1925, cxxi, 1.

It is of interest that André Thomas established in his studies on inducing angiospasm the following symptoms: cramps, numbness, decrease of the local temperature and disappearance of pulsations. Our findings in some of these cases coincided with his. Exercise does undoubtedly cause spasm—due partly, at least, to the fact that the amount of blood supplied does not correspond to the demand for the work performed.

Intermittent claudication associated with arteriosclerosis frequently appears between the ages of twenty and forty and is of toxic origin, resulting from rheumatism, syphilis or nicotine. The blood-pressure readings were subnormal, a fact which is in agreement with the observations of Bernheim.

CASE REPORTS. CASE I.—W. F., aged sixty years, admitted to the Episcopal Hospital, April 11, 1922. His chief complaint was numbness, which began the previous day while putting in a gaspipe. He noted a tingling sensation in the left arm and left leg, quit work, walked a few squares and went to bed. Soon there developed a total paralysis of the left leg and partial involvement of the left arm, and the patient was sent in by his family physician. His past history was negative, save for pneumonia at twenty. The patient was a laborer, and denied the use of either alcohol or tobacco.

His father had died at sixty-five with a similar attack, his involvement, however, having been the right arm and right leg and having lasted five weeks. At forty his nephew had a similar attack, lasting three or four months, the patient was unable to recall the extremities involved. His mother died at sixty-five of “softening of the brain.”

Interesting features of his physical examination were: heart, hypertrophied to nipple line; weak muscular tone with accentuated pulmonic second sound; paralysis of left leg and left arm, except that there was a slight movement of the fingers. There were no retinal hemorrhages.

BLOOD PRESSURE			
	S.	D.	Arm.
On admission	130	75	Right
	110	55	Left
April 19, 1922	120	55	Right
	145	70	Left
April 21, 1922	135	55	Right
	130	45	Left
April 27, 1922	140	55	Right
	130	60	Left

During his stay in the hospital he responded promptly to external heat and supportive treatment. Motion slowly returned and he was up and around the ward several days before his discharge. The patient was discharged April 29, 1922.

CASE II.—W. V. C., aged sixty years, admitted to the Episcopal Hospital May 24, 1922. His chief complaint was weakness, followed by partial facial paralysis. It began on the morning of the day of admission, while he was working. The first thing he noticed was pain, with partial paralysis of the right side of his face, facial movements being very weak. He was sent to the receiving ward immediately with the partial paralysis noted above and also weakness of the muscles of both arms. He also complained of headache and muttered incoherently. Aside from that his symptoms were negative, as were his previous medical history and family history. His social history is that he was an electrical-assembling worker; however, he never worked in lead. The salient features of his physical examination were slight hypertrophy of the heart just within the nipple line, the apex-beat being in the fifth interspace. The muscular tone was noted as being fair. The ward notes, dated the day following admission, state that there was no noticeable weakness in any group of muscles. The eyes showed a slight tendency to oscillatory and lateral nystagmus. The left pupil did not close tightly.

On admission, his blood-pressure was 135 systolic, 75 diastolic; which arm used was not stated; however, on the day after admission it was 130 systolic and 75 diastolic in both arms. On admission his temperature was 99.3° ; pulse, 80; respiration, 20. During the six days he was in the hospital his temperature ranged between that on admission and 97° , while the pulse-rate was between 60 and 72. All laboratory findings in this case were negative. The patient was discharged June 6, 1922, in extremely good condition.

CASE III.—J. McC., aged sixty-four years, admitted to the Episcopal Hospital March 24, 1922. Complained of pain in left chest, pain and weakness in the left arm, and the left foot was swollen from the ankle to the knee. The history of the present illness, as noted in the hospital records, is entirely barren of any symptoms having a bearing upon the subject of vascular spasm with the exception of the fact that the patient noticed weakness in his left arm. Therefore, the deductions are made from our findings. The social, previous medical and family histories also were negative and valueless as far as our records are concerned.

The physical examination showed the teeth to be in extremely bad condition. The heart was enlarged and muscle tone weak. The eyes showed a slight dilatation of the right pupil. The blood Wassermann was negative. The urine was typically that of an arteriosclerotic, namely, a trace of albumin, low specific gravity and an occasional hyaline cast. Phthalein elimination was 30 per cent for the first two hours. Blood-urea nitrogen and differential blood count were negative. Red blood cells, 3,630,000; white blood cells, 21,920. The spinal-fluid examination was negative, except that the fluid was under increased pressure. Dr. Kraus, the ophthalmologist, states the disk of the eye was very gray and there was a pulsation of sclerotic vessels. The temperature in the left popliteal space was 97.2° , while that in the right was 95.2° . This difference was noted until May 8, after which the temperature became even. Before the latter event there was considerable edema in the left leg. After some improvement from the treatment indicated as a result of his examinations he was discharged May 19, 1922, our feeling having been that our data and findings were sufficient to warrant his being in the classification of cases mentioned in this paper.

CASE IV.—F. B., aged sixty-seven years, admitted April 6, 1922, to the men's medical ward, Episcopal Hospital, with a diagnosis of subdural hemorrhage. The hospital records state that the history of the present illness, and in fact all history, was unobtainable on admission, as the patient was unable to talk, yet understood all commands.

The physical examination showed his teeth to be in extremely poor condition and there was also a pulsation in his neck. The heart was normal in outline and there were no thrills, murmurs or shocks. The abdomen and extremities were negative. The eyes reacted normally to light, but there was a nystagmus to the left and also slight asymmetry to the left. Patellar reflexes were exaggerated. Suggestive Babinski reflex on the left side. The spinal-fluid examination was negative, except that the fluid was under moderately increased pressure. The eye examination by Dr. Fewell, the ophthalmologist, was as follows: Pupils dilated—right 6 mm., left 3; color, well defined; arteries seem reduced in caliber, showing a moderate sclerosis; no discreet lesions in the fundus. O. S. the same. The urine and blood examinations were negative. The blood-pressure was 150 systolic, 95 diastolic, in both arms.

The temperature while in the hospital varied between 100° first few days to normal. The pulse was 96 on admission, but soon became normal. The patient was treated with heat and stimulation and his condition improved steadily, so that at the time of his discharge, May 27, he was able to walk around the ward and seemed to have regained the complete use of all the disabled members.

CASE V.— M. M., admitted to the men's medical ward of the Episcopal Hospital June 28, 1923, with a diagnosis of intermittent claudication. Patient stated that while walking down the street, three weeks before admission, he suddenly felt a sharp pain in the calf of his right leg; so sharp, in fact, that he thought something had struck him. The weakness and pain became more marked and he went home and then to bed. The leg became swollen and extremely warm from the knee-joint to the ankle and was sore for about two weeks. The condition improved and he was able to be up and around, but could walk a short distance only, suffering no pain at that time. However, two days before his admission he had another attack of pain similar to the previous one and the leg became swollen again. There were no other cardiovascular symptoms and the remainder of the history, both as far as his family and social histories were concerned, was negative. Previous medical history was also negative as far as acute infections were concerned. He had been a moderate tobacco-user, but denied having ever used alcohol.

Physical examination was negative, except that the heart sounds were distinct and of fair quality. The right leg from the knee down showed edema and ecchymosis and pitted on pressure. There was also slight tenderness on deep pressure on the calf of it. The temperature of both the right and left leg was identical and both anterior and posterior tibial arteries pulsated. The blood-pressure was not recorded. Pulse-rate during his stay in the hospital varied between 84 and 76, while his temperature was practically normal. Urinalysis and blood count were negligible. The ward notes state that by July 2 the edema in the right leg had decreased markedly and the yellowish discoloration was disappearing. After application of dry heat his condition improved so markedly that he insisted upon being discharged on July 5, contrary to medical advice.

CASE VI.—J. B., aged fifty-two years, admitted to the men's medical ward of the Episcopal Hospital July 6, 1923. The history was of his having fallen unconscious in a trolley car while on his way

to work. Before that time he suffered from headache and mental confusion. While standing on the platform of an elevated car he felt dizzy, had a sharp frontal headache and then became unconscious and remained so until he was brought to the receiving ward of this hospital. He stated that he had had several similar attacks within the last few years, during which he did not void urine, did not froth at the mouth, did not have convulsive movements and did not bite his tongue.

The patient was a police-officer. He drank four cups of coffee a day, with an occasional drink of whiskey.

Physical examination showed that the heart was enlarged to the left and the muscular tone was fair. There was a definite sclerosis of the upper vessels of the arm. Laboratory reports were entirely negative. Blood-pressure was 124 systolic, 65 diastolic.

This condition improved while resting in bed and we were unable to persuade him to stay in the hospital longer than the eleventh of April.

TREATMENT. The treatment of vascular spasm consists of complete rest in bed, liquid or semisolid diet, continuous application of heat to involved areas and supportive medication.

In arteriosclerotic patients, potassium iodide is indicated, while in many cases tincture of belladonna is advised; Morichau Beauchant has advised the use of sodium citrate, 3 to 4 gm., over long periods.

SUMMARY. 1. Vascular spasm is rare.

2. Intermittent claudication is a local example of the condition.

3. It must be borne in mind when any hemiplegia, monoplegia, aphasia or unilateral edema is seen.

BIBLIOGRAPHY

Allbutt, Sir Clifford: *System of Medicine*, by many authors, McMillan & Co., Ltd., London, 1909, vi, 571.

Beauchant, R. Morichau: *Treatment of Vascular Spasm*, *Bulletin de la Société médicale des hôpitaux*, Paris, March 3, 1922, p. 404.

Bernham, Bertram M.: *Blood-pressure Findings in Circulatory Disorders of the Extremities*, *Journal Am. Med. Assn.*, March 18, 1922.

Cabot, Richard C.: *Differential Diagnosis*, Vol. II, W. B. Saunders Company, Philadelphia, Pa. and London, 1918, p. 469. Case 196.

Campbell, D. J.: *Arteriosclerosis and Angina Pectoris with Temporary Muscular Paralysis—Case Report*, *Canada Med. Assn. Jour.*, Montreal, February 1922.

Cruschmann, Hans: Diagnosis and Treatment of Intermittent Claudication, Münch. med. Wchnschr., November 11, 1921.

Ernst, Tobias: A Case of Early Intermittent Claudication Umarteriosclerotic and Accompanied by Manifestations of Raynaud's Disease, Berl. klin. Wchnschr., December 18, 1921, lviii, 1493.

Osler, Sir William: Principles and Practice of Medicine, Eight Edition, D. Appleton & Co., New York and London, 1918, p. 846.

Pavlovsky, A.: Edema of Arm, Acute or Subacute, Following Exertion, Semaine méd., vol. xxviii, p. 317.

Thomas, André: Induced Angiospasm in Peripheral Arteritis and Intermittent Claudication, Presse méd., Paris, December 6, 1922, p. 1049.

Zak, E.: Vascular Spasm in Intermittent Claudication, Wiener Archiv. für innere Medizin, Vienna, June 30, 1921, p. 4012; Spasm of Vessels in Intermittent Claudication, Med. Klinik, Vienna, April 8, 1923.

OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY¹

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THE symptomatology of occlusion of the posterior inferior cerebellar artery was first firmly established by Wallenberg¹ in 1895; since then many clinical cases and a few with necropsy have been recorded. Hun and Van Gieson,² in 1897, and Spiller,³ in 1908, were the first Americans to report cases with necropsy. Goldstein and Baumm,⁴ in 1913, reviewed the cases reported up to that time, tabulated the symptoms and concluded from their statistical study that the most constant clinical sign was a loss of pain, heat and cold in the area of the fifth nerve on the side of the lesion and on the opposite side of the body. Since 1913, only an occasional article has been written on the subject; one of the most recent and important is the one by Foix, Hillemand and Schalit.⁵ These authors consider the vascular distribution of the medulla, as follows: (1) The paramedian artery, supplying a triangular area which includes the pyramid, fillet and part of the olive. (2) The artery of the lateral fossa of the bulb, which nourishes the lateral portion of the medulla except the restiform body, which is supplied by (3) the inferior cerebellar artery. They believe that the "artery of the lateral fossa of the bulb" is the one that is usually involved in the "syndrome of the posterior inferior cerebellar artery occlusion," and not the inferior cerebellar artery itself; but, in one of our patients (Case II),

¹ From the Neurological Department of the School of Medicine of the University of Pennsylvania, the Episcopal Hospital and the Laboratory of Neuropathology of the Philadelphia General Hospital. Reprinted from *The Journal of Nervous and Mental Diseases*, 1927, lxx, 125.

the restiform body was also involved in the softening, and this case at least does not fit in with the rigid classification given by the French authors. It is true that clinically there would be no difference in the symptomatology, since the same cerebellar symptoms could occur from involvement of the cerebellar tracts themselves prior to their entrance into the cerebellar peduncles.

The following cases (one with necropsy) are herewith reported:

CASE I.—A white male, aged fifty-four years, with a negative family history, had smallpox in 1871 but denied syphilis. He drank as much as a quart of whisky a day. On September 28, 1918, he suddenly felt a sharp pain in the right occipital region, which crossed to the left side of the head and radiated down the left arm and leg. He became unsteady on his feet, but managed to get home without assistance, although he was very dizzy. He developed difficulty in swallowing and his voice was reduced to a whisper. He did not see double and did not vomit. He stayed in bed four weeks because he staggered like a drunken man. The entire left side of the body felt numb and dead, and at times he had the sensation of pins and needles there. The face was not involved in this paresthetic phenomenon.

At the end of a month he left his bed for the first time, although he still had a tendency to stagger when walking. He noticed, when he touched a hot object with the left hand, it did not give him the sensation of heat, but of tingling. He had been very drowsy since the onset of his trouble. He never noticed any difference in the amount of perspiration on the two sides of his body.

Examination. He was seen on June 2, 1919, nine months after the acute attack. He walked with the base of support widened and he had a tendency to stagger to both sides. He had a distinct sway with the eyes open; when the eyes were closed he deviated to the left, although he then actually fell to the right. The right pupil was smaller; both were irregular and reacted sluggishly to light. The right palpebral fissure was narrower than the left. The eye grounds were normal; the vision was 6/25 in the right eye and 6/200 in the left. The ocular, fifth and seventh nerves were normal. The uvula, when moved, was drawn upward and to the left; stimulation of the pharynx on the left produced gagging, and on the right no response. The right vocal cord was paralyzed.

The Bárány tests (performed by Dr. Lewis Fisher), summarized,

were as follows: The vestibulo-ocular tract from the horizontal canal on the right side showed slight evidence of disturbance, because the nystagmus produced was of the normal horizontal movement, but was mixed with an oblique movement. The vestibulo-cerebello-cerebral tract for vertigo from the horizontal canal did not function at all, thus indicating that something had interfered with it after it had become separated from the vestibulo-ocular tract, most likely in the right restiform body. The absence of vertigo and practically no nystagmus from the vertical semi-circular canals suggested an interference with the vertigo as well as in the nystagmus tracts from these canals. Dr. Fisher concluded from his examinations that the disturbance was due to interference with the circulation involving the brain-stem on the right side about its middle.

Muscular power was good in all four extremities. All the deep reflexes were active and equal on the two sides with the exception of the Achilles reflexes which were lost. Plantar stimulation produced flexion of the toes on both sides. Slight adiadokokinesis was noted in the right upper extremity.

Sensation: stereognosis, vibratory sense, sense of position and touch were normal throughout; but pain, heat and cold were diminished, but not lost, on the left side of the body. Sensation was not affected on either side of the face and the corneal reflexes were normal. The blood Wassermann, urine, blood-pressure and heart were normal.

The man made a good recovery, but at the time of his last examination still had the clinical evidence of his lesion.

CASE II.—A white woman, aged forty-eight years, was admitted to the Episcopal Hospital on March 15, 1924. Her husband had died two years previously and had suffered for some years before his death from tabes dorsalis. Ten years before her admission the patient had a generalized skin eruption, presumably due to syphilis. Her chief complaint was inability to swallow. About one month before she entered the hospital, she suddenly developed numbness and weakness in the right arm and leg and thickness in speech. In three or four days she entirely recovered and returned to work. Three days before admission she suddenly developed difficulty in swallowing, numbness of the left side of the face and difficulty in speaking.

Examination. Distinct narrowing of the left palpebral fissure was present, due to enophthalmos. The left pupil was smaller than

the right and was irregular; both pupils reacted well. The eyeballs could be moved freely in all directions. The corneal reflex was lost on the left side. The nasolabial fold on the left was deepened and suggested the condition seen in an old facial palsy; however, the movements of the face were normal on both sides, and no changes in the electrical reactions were present. Saliva continually ran from the mouth and great difficulty in swallowing was noted. She could barely whistle, but could blow out a match at a fair distance from the mouth. The tongue was protruded in the midline. The patient was too ill to stand or walk. Both patellar reflexes were present; the right was exaggerated. Plantar stimulation produced an atypical Babinski on the right and flexion of the toes on the left.

Sensation: A marked impairment of pain, heat and cold was demonstrable in the distribution of the left fifth nerve and on the right side of the body. Sense of position and stereognosis were normal. An ataxic tremor was present in the finger-to-nose test on both sides.

Laboratory examination: The blood Wassermann test was +2 in the cholesterinized antigen. The routine blood examination was normal and her urine showed the findings of nephritis. A bloody spinal fluid was obtained, due to local injury.

Clinical Course and Outcome. The patient lived twelve days after admission to the hospital. In this time the tongue protruded to the right and the temperature of the right side of the face was definitely higher than on the left. She developed retention of urine and later incontinence. She died suddenly.

Pathology. The brain on gross examination showed moderate convolutional atrophy, haziness of the piaärachnoid and intense plaque formation of the vessels, although no thrombosis could be made out. Microscopic examination: A serial-section study was made of the brain-stem and midbrain. On the left side of the medulla was an area of softening which was quite recent and occupied the region of the restiform body and all structures anterior to it up to the inferior olive. The lesion was widest at about the middle of the medulla and extended upward and downward in a cone shape. Other small areas of softening were found, one involved the left superior cerebellar peduncle, another and larger area was found in the left middle cerebellar peduncle and in the left lenticulo-capsular region. The pyramidal system showed no degeneration by the Weigert or Marchi methods. The vessels uniformly were thickened, especially the smaller ones.

COMMENT. We believe that the two cases reported above are instances of occlusion of the posterior inferior cerebellar artery. The one with necropsy undoubtedly had this condition, and in addition had other areas of softening in the cerebrum and brain-stem, the condition being syphilitic in origin. The first case was unusual in that there was no clinical evidence of implication of the descending root of the fifth nerve. The involvement of the descending root of the fifth nerve in the medulla is one of the commonest signs in this condition; however, other cranial nerves were involved and helped to make the localization possible in this case.

BIBLIOGRAPHY

1. Wallenberg: Arch. f. Psych., 1895, xxvii, 504; Arch. v. Psych., 1901, xxxiv, 923; Deutsch. Zeitschr. f. Nervenheilk., 1911, xli, 8; Deutsch. Zeitschr. f. Nervenheilk., 1901, xix, 227.
2. Hun and Van Gieson: New York Med. Jour., 1897, lxv, 513, 581, 613.
3. Spiller: Jour. of Nerv. and Ment. Dis., 1908, xxxv, 365.
4. Goldstein and Baumm: Arch. f. Psychiat., 1913, lii, 335.
5. Foix, Hillemand and Schalit: Rev. Neuro., February, 1925 Pt. I, No. 2, p. 160.

CLINICAL STUDIES OF LETHARGIC ENCEPHALITIS*

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WHEN we first heard of lethargic encephalitis, associating and connecting it with the influenza epidemic, our impression was that we were dealing with a new disease. This proved, however, to be erroneous, for, as a matter of fact, Camerarius mentioned it under its present popular name of sleeping sickness in connection with his description of an influenza epidemic in Tübingen in 1718. Although the definite relationship between encephalitis lethargica and epidemic influenza is still an open question, in favor of those upholding the opinion that the disease is distinctly related to influenza is the fact (which is undoubtedly most suggestive) that it has appeared in epidemic form only when influenza has been epidemic. The opponents of that view point to the fact that in less than 50 per cent of the cases reported in the literature is there a history of influenza.

BACTERIOLOGY. As yet there has been no positive identification of the causative organism, that term being used advisedly and meaning to establish a bacillus isolated and recognized by other workers. Most encouraging, however, is the recent work of Levaditi and Harvier, who made intracerebral injection of emulsions of brain from patients dead from lethargic encephalitis and thereby produced lesions in rabbits similar to those seen in man, and then a later transference of the disease to both guinea-pigs and monkeys. Further important information brought out by them is the fact that the virus in emulsions of rabbit-brain tissue would pass through Chamberland filters, Nos. 1 and 3. Inasmuch as 4 of the 6 rabbits injected with the filtrate contracted the disease, while all 6 controls became sick, it appears that fil-

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tration in some way reduces the activity of the virus. An equally important addition to our knowledge is the result of the work of Loewe and Strauss in their reports that Berkefeld filtrates of brain material, nasopharyngeal mucous membrane and nasal washings from cases of epidemic encephalitis have produced in rabbits and monkeys lesions typical of this disease. Spinal fluid and blood have also produced the disease experimentally in these animals. The virus has been passed through many series of animals.

Suggestive at least is the laboratory conclusion here reached, viz., that this disease is transmitted by secretions of the nasopharynx and that mild and abortive cases and carriers play an important part, when it corroborates the clinical findings of William Boyd¹ in an epidemic of the disease in Winnipeg, Manitoba.

PATHOLOGY. The most striking thing about the brain of a patient who has died from lethargic encephalitis is the marked degree of congestion and edema, which may be either local or general. Although in some cases the entire cerebrum was involved, the brain-stem was most frequently affected. Small pinpoint hemorrhages were frequently seen in the medulla, while secretions showed a perivascular round-cell infiltration. This latter was noted most often near the fissure of Sylvius and the floor of the fourth ventricle, extending into the cord and sometimes into the basal ganglia. Grossly, many brains show nothing pathological, yet these same brains show microscopically a round-cell infiltration.

SYMPTOMATOLOGY. The polymorphism of this disease gives us a wide and varied symptomatology. This has resulted in the appearance in the literature of many classifications, broad, comprehensive and inclusive, especially that of Roger, yet not as practicable for the internist as that of MacNulty, which is as follows:

1. Cases with general symptoms and without localizing signs.
2. Cases with third-nerve paralysis and general disturbance in the function of the central nervous system.
3. Cases with facial paralysis and general disturbance in the function of the central nervous system.

4. Cases with spinal manifestations and general disturbance in the function of the central nervous system.

5. Cases with polyneuritic manifestations and general disturbance in the function of the central nervous system.

6. Cases with mild or transient manifestations (so-called abortive cases).

The symptoms are both general and localizing, the former including lethargy, fever, headache, body pains and coated tongue, while the latter are referable to the cranial nerve or nerves involved. To my mind, the outstanding symptoms are lethargy and a negative spinal fluid. In cases where the third- or sixth-nerve nuclei are involved, of course, ocular symptoms are prominent. A diagnosis is not easy, and in order to make it we must make use of every means at our command, viz., careful history and physical examination and thorough laboratory tests done by a well-trained, competent technician.

It has been my fortune to study the cases reported herewith and I have been enabled to state the present condition of the living patients through the coöperation of the Social Service Department of the Episcopal Hospital.

CASE I.—J. G., aged forty-five years, was admitted to the Episcopal Hospital April 15, 1920. The family history was negative. His previous medical history was negative until two months before admission, when a cough started, accompanied by a slight mucoid expectoration and a loss of weight and strength. During that time he also felt indisposed for his work as grocer, and was rather tired and sleepy. About four weeks before admission to the hospital the weariness and sleepiness increased, and two weeks later he was unable to continue with his work but went to bed complaining of exhaustion. Following this, his wife noticed that he began acting queerly and talking in a rather peculiar way. He refused to remain in bed, became most irrational at night, sleeping very little, and finally, the day before admission, he went into a restless, muttering delirium. Examination on admission showed a rather anemic man with masked facies and a toxic appearance. He was restless and refused to stay in bed, had slight difficulty in speech, talked incoherently to himself, though he would answer questions intelligently, drifting back into mental confusion.

The positive physical findings were as follows: Pupils contracted and reacted sluggishly to light; marked tremor of the tongue; general muscular hypertonia, especially of the neck, but no actual rigidity; abdominal reflexes normal but patellar exaggerated; a positive Kernig's sign on both sides.

The laboratory findings were: Urine: specific gravity, 1020; albumin, cloud; sugar, negative; light and dark granular casts; no red blood corpuscles; occasional white blood corpuscles. Blood: hemoglobin, 65 per cent; red blood corpuscles, 4,300,000; white blood corpuscles, 11,600; differential: polynuclears, 62 per cent; mononuclears, 12 per cent; small mononuclears, 24 per cent; basophiles, 2 per cent. Wassermann, positive, 3 +. Spinal fluid, not under pressure, clear; 91 cells to the c.mm.; polynuclears, 90; lymphocytes, 10; Fehling's reduced; globulin, negative.

After three days, from a condition of restless muttering delirium (in which restraint was necessary) he lapsed into a deep coma. He lay with mouth open, eyes closed and face immobile and did not respond to questions. During the three days he ran a hectic temperature, ranging from 97° to 103° F. (rectal). He took liquids well and his excretory functions were normal. During the next three days he became steadily worse, ptosis of both eyelids developed, with paralysis of the soft palate and dropping of the lower jaw, and finally death occurred at 5 A.M. April 21.

REMARKS. The interesting features in this case are the absence of any apparent influenzal infection aside from slight bronchial irritation, the absence of the more usual initial symptoms, such as diplopia and ptosis, and finally the severe maniacal development which ensued. The prodromal period may be regarded as having existed possibly for a period of two months. Fever may or may not have been present during the prodromal period, but while the patient was in the hospital he ran a hectic temperature, ranging from 96.4° to 106° F., the latter being terminal. The presence of a Kernig's sign is of particular interest in that it is usually described as being lacking in this condition. We must also note the rather high cell-count in the spinal fluid, although it is by no means uncommon to find a count as high as this.

CASE II.—C. F., aged forty-three years, was admitted to the Episcopal Hospital, April 16, 1920. The family history was

negative. The personal history was negative until four weeks before admission when he had influenza: this was followed by rhinitis and pain in the frontal sinus and in the left ear. These cleared up in a few days and he felt well until the beginning of the present illness, which began one week before admission, with pain in the left shoulder, the muscles of the chest and in the gastric region. Three days later he manifested tremors of both hands and twitching of the abdominal and lower intercostal muscles, associated with shooting pains over the same region. These muscular contractions and pains prevented his sleeping for the three nights previous to admission.

Examination on admission showed a masked expression but no visual disturbances or palsies; slight tremor of both hands; heart and lungs negative; abdomen distended and the upper recti muscles slightly rigid; some tenderness to pressure over the epigastrium. There was also a constant clonic contraction of the upper abdominal muscles, the lower intercostals and apparently the diaphragm. He had considerable belching but no hiccough, nausea or vomiting. Abdominal and patellar reflexes were slightly exaggerated. Mental condition was lucid.

The laboratory findings were: Urine: specific gravity, 1012; albumin, very faint trace; sugar, negative; no casts, red blood corpuscles or white blood corpuscles; many motile bacteria. Blood: white blood corpuscles 16,000; Wassermann, negative.

Patient's Condition While in Hospital. He talked a great deal during the night, got out of bed and seemed confused when put back. During the following five days he was irrational at times, had delusions and hallucinations and slept but little. The abdominal twitching was constant. His temperature ranged from 100° to 103° F. From this state of delirium, in which restraint was necessary, he became lethargic: however, he could be aroused and answered questions rationally. At the end of five days more, although he slept a great deal, there was marked improvement; his expression became more natural and for the first time the abdominal twitchings were absent. He complained of a tingling and numbness of the skin and shooting pains in the chest and abdomen. These gradually disappeared, as did all tremors, but lethargy was marked for a week following, during which time the patient often fell asleep while sitting up.

REMARKS. This case clearly followed an influenzal infection of recent origin. The patient's symptoms were ushered

in by pain in the shoulders, chest and gastric region, and tremors of the hands and abdominal muscles; hence he falls more within the polyneuritic classification, though the pronounced maniacal tendency rather precludes a clear-cut classification. His most constant symptom was the spasmodic contractions of the muscles of the upper abdomen, a symptom regarded by some as pathognomonic of lethargic encephalitis.

In spite of repeated requests this man did not return to the hospital for an examination by me. The social service reports that he is working, but not at his old occupation. Since discharge from the hospital he has been under the care of a doctor and is much improved.

CASE III.—S. B., male, aged fifty-two years, was admitted to the Episcopal Hospital on April 14, 1920. His family history was negative. The personal history revealed the fact that for about two months before admission he suffered markedly from nervousness, which he felt was due to the strain of his work in the fuel-oil plant at the Navy Yard; he also had gaseous eructations and epigastric pain. In addition, he had pains in the muscles of the legs, with tingling and numbness. On admission to the hospital, the patient showed a continuation of the above-mentioned symptoms, plus diplopia. Tremor of the legs became persistent and violent and he was unable to sleep.

Examination showed an extremely restless and nervous man, who had slight difficulty in speech, which was stuttering and scanning. The facies was masked. There was a suggestive bilateral ptosis of eyelids: the pupils were contracted and equal, responding but slightly to light and accommodation. The tongue was tremulous and deviated toward the right. There was no abnormality of either the heart or lungs. There was muscular resistance of the abdomen and the reflexes were present and a tache noted. The tremors of the legs were convulsive: the patellar reflexes could not be elicited at this time. Ankle clonus was marked on the right side: the Babinski reflex was negative while Kernig's sign was suggestive. There were marked tremors in both hands.

Laboratory findings were: Urine: specific gravity, 1012; albumin, trace; sugar, negative; occasional granular and hyaline cast; few red blood corpuscles; few white blood corpuscles. Blood: hemoglobin, 70 per cent; leukocytes varied between 11,000 and 14,000; the differential count was normal, with polymorphonuclears

predominating. Wassermann test was negative. Spinal fluid, under pressure: clear; 45 cells to the c.mm.; Fehling's solution was reduced; globulin was negative; no organisms were found on the smear.

Patient's Condition While in Hospital. He went almost immediately into a muttering delirium and became somewhat resistive, rendering restraint necessary. He slept some but muttered almost constantly. After three days of restless delirium, during which time his temperature rose from 101° to 104° F., he went into a profound lethargic state and could be aroused only with difficulty. The tremors disappeared at this time, but the patellar reflexes were present. Marked bilateral ptosis, difficulty in speech, masked facies and lethargy were most pronounced at this time. During the following ten days there was a gradual decline in temperature from 104° to normal, accompanied by a slight improvement in both mental and general condition. He slept quietly most of the time, but occasionally got out of bed and seemed disoriented. All of his symptoms slowly disappeared and he was discharged much improved late in July. The classification of this case is a difficult one, but should lie between the polyneuritic and psychotic type.

Condition of Patient one Year Later. He returned to work in September, 1920; his position required great accuracy and mathematics. At first he was a little shaky, a sensation which he experienced for about three months. He was still very nervous and had difficulty in getting to sleep: it usually took about three hours. He had no mental depression nor any difficulty in concentrating. There had been no eye symptoms and very few headaches, nor had he suffered from any abdominal pains. He looked well and had gained 50 pounds. His eyes reacted normally to light and accommodation; his knee jerks were normal. There was a slight tremor of the right hand and a very slight ataxia.

CASE IV.—W. T., male, aged thirty-two years, was admitted to the Episcopal Hospital, April 6, 1920. His family history was negative. His personal history was also negative until two months before admission, when he had an attack of influenza which confined him to bed for one week. Following this he had a frontal-sinus infection which cleared up in a few days.

Present illness began three weeks before admission when, after feeling unusually well, he awoke one morning with diplopia. This continued for one week, when he consulted an eye specialist who dilated and refracted the eyes: the diplopia continued, however, and a purulent conjunctivitis developed. One week before admis-

sion he went to bed, complaining of his eyes and of being drowsy. Four days before admission he began having difficulty in articulation, which has increased until he could not speak. The day before admission he had difficulty in swallowing. Physical examination showed an expressionless face, with tremor of the muscles around the mouth. The pupils were dilated and did not react to light: there was a bilateral ptosis and a catarrhal conjunctivitis. The tongue was protruded with difficulty and was tremulous and coated. There was resistance in the muscles of the neck, but no rigidity. He could not talk, although it was evident he understood what was said to him, as he endeavored to do what he was told. Heart, lungs and abdomen were negative. Patellar reflexes were exaggerated: there was no Babinski or ankle clonus: Kernig's sign was suggestive. A general muscular hypertonicity was noted.

Laboratory findings were: Urine: specific gravity, 1.017; albumin, negative; sugar, negative; no casts, no red blood corpuscles. Blood: hemoglobin, 80 per cent; red blood corpuscles, 4,150,000; white blood corpuscles, 9400; differential, normal. Wassermann, negative. Spinal fluid, not under pressure; 23 cells to the c.mm.; Fehling's reduced; globulin, negative. Eye examination: Slight edema of the retina; slight swelling of the fundus; vessels engorged.

Patient's Condition While in Hospital. He swallowed liquids fairly well. The facies remained masked and the muscular resistance general: the twitching of the mouth muscles was present continuously. At the end of the first week the conjunctivitis had cleared up, he began to talk slowly and showed general improvement. At the end of three weeks he could articulate distinctly and felt well, although the tremors were still persistent. The day following this report of his condition, at the end of three weeks, he had a prolonged visit from some of his friends and his difficulty in speech and swallowing became very marked; furthermore, he stared at one object constantly, with his eyes wide open. He reacted slowly to any stimulus; he did, however, complain of a tingling and numbness of the third and fourth fingers of each hand; his sleep was disturbed by dreams and for the first time he became solicitous about his condition. At the end of a few days he showed a general improvement, though there was a slight paralysis of the left facial nerve. Although lethargic in appearance, he slept but little. His condition continued to improve slowly and he insisted upon leaving the hospital late in May.

REMARKS. This case was typical of the many reported in the literature and offered no difficulty whatever from the

viewpoint of diagnosis. Report of present condition shows that he feels well except for weakness in both feet and legs: he also tires easily. He has trouble getting to sleep and often awakens during the night. At times he becomes confused, especially by excitement: he is much slower to form ideas than formerly. His wife states that his disposition has changed very much and he is solicitous about his condition. He feels little stiffness in the muscles of his neck and there is a slight weakness of the muscles of the left side of his face. He has been doing clerical work since October 1, 1920. His patellar reflexes are exaggerated on both sides. His left pupil reacts slightly more promptly than the right. There is no ataxia.

CASE V.—C. L., male, aged twenty-two years, was admitted to the Episcopal Hospital, April 30, 1920. His family history was negative. Personal history showed that twelve weeks previous to admission he had a severe attack of influenza, followed by double pneumonia and pains of a rheumatic character in the shoulders and legs. He was confined to bed for eight weeks. During his convalescence in the two following weeks he suffered from a severe cough with slight expectoration. During this time he complained of weakness, exhaustion and dizziness and slept a great deal, mentioning in addition the pains in his shoulders and legs. Two weeks before admission he returned to his work of driving a milk wagon and at the time complained of being sleepy and unable to wake up in spite of sleeping thirteen hours a day. During this period he seemed rather irritable and his speech was unnatural. The present illness began four days ago. While driving his milk wagon he suddenly lost the power of speech, complained of diplopia and inability to change money correctly. He drove back to his home, where he collapsed and thought he had a stroke. His expression was unnatural and he had general tremors, which were most pronounced in the legs. Immediately following this he was unable to move his left side and his speech did not return.

Physical examination showed a well-developed man with a masked expression, markedly jaundiced and unable to talk or move the left arm or leg. His eyes reacted normally to light and accommodation; there was no nystagmus or ocular palsies; the sclera were slightly icteric. The mouth was pulled to the right and he was unable to move the facial muscles of the left side or wrinkle his forehead. There was a slight tremor of the tongue. As was

the case with W. T., it was evident that he understood what was said to him, as he endeavored to do what he was told. The neck showed a slight muscular resistance. The chest was negative except for a few moist râles at the base of the right lung. The heart showed forcible pulsation against the chest wall, a precordial thrill and a blowing systolic murmur heard most distinctly over the pulmonic area. The first sound was loud and roughened: the quality was poor and varied a great deal over the different areas, and there was marked arrhythmia. The abdomen was markedly jaundiced and there was slight tenderness over the gall-bladder region, together with a muscular resistance of the upper abdomen. The reflexes on the right side were normal; a tache was obtained. The patellar reflexes were present and exaggerated on the left side where the Babinski was present; Kernig's sign was suggestive on both sides; there was a flaccid paralysis of the left arm and leg.

The laboratory findings were: Urine: specific gravity, 1014; albumin, slight cloud; sugar, negative; many granular casts; few red blood corpuscles; few white blood corpuscles. Blood: white blood corpuscles, 14,800; Wassermann, negative. Spinal fluid, not under pressure; 26 cells to the c.mm.; does not reduce Fehling's; globulin, negative.

Patient's Condition While in Hospital. During the first week he began to articulate fairly well speaking short sentences. The facial paralysis of the left side naturally changed but little. His appetite was good and he slept quite normally. His temperature dropped to 98.3° and his mental condition was clear. The jaundice began to clear up rapidly and he complained of but few epigastric pains. By the end of the following ten days, power in his left hand began to be shown and there was fairly good motion in his left leg. The jaundice also cleared up and his condition was highly satisfactory. He was discharged on the first of June as improved.

Patient's Condition One Year Later. This patient disregarded my request to present himself for examination; however, the Social Service Department learned that he had had treatment at the Nervous Dispensary at the University Hospital, so that from the notes at the time of his last visit, which was late in March, I am able to give the following report: He had a distinct amnesic aphasia, evidently an aftermath of his lethargic encephalitis. He could understand all spoken phrases fairly well, but complicated or difficult ones seemed to confuse him. He had no word blindness or word deafness and no signs of a hemiplegia. He had no hemianopsia. The patellar reflexes were exaggerated: the eyes reacted normally to light and accommodation.

CASE VI.—T. B., aged thirty-four years, an Italian, was admitted to the Episcopal Hospital, May 10, 1920. His family history was negative. His personal history was also negative until March 10, 1920, when he had an attack of influenza which confined him to bed for about two weeks. Following this attack he had a pain in the right ear and over the right eye, which passed off in a few days, although the hearing in his right ear had been impaired ever since. He felt well then until two weeks previous to admission. His present history began at that time with a feeling of illness but no definite complaint, simply a malaise and anorexia. In a few days he began having twitching of the muscles of the right side of his neck, which became associated with sharp pains in the neck and shoulders, extending down the right arm. The pain and the constant muscular twitching kept him awake at night. Six days before admission he began to have tremors of both hands and a diplopia which lasted two days. Then he noticed difficulty in articulation. At the time of his admission he complained most of the twitching of the muscles of the neck, pain in the neck and right arm, and inability to sleep.

The physical examination showed a masked facial expression with head retracted backward and slightly to the right side. The pupils were dilated, but responded to both light and accommodation. There was a marked tendency to stare. In the right ear was noted a slight impairment of hearing. The tongue was not tremulous. The neck showed constant convulsive twitching of the trapezius muscle most marked in the cleido-occipitalis portion. There was the painful sensation of an ordinary neuritis confined to the nape of the neck, shoulder and right arm and hand. Marked muscular resistance of neck was noted. The heart, lungs and abdomen were negative. The patellar reflexes were increased; Kernig's sign was absent; there was no ankle clonus and no Babinski.

The laboratory findings were: Blood: hemoglobin, 75 per cent.; red blood corpuscles, 5,890,000; white blood corpuscles, 8200; differential showed polymorphonuclears 72; lymphocytes, 28. Wassermann, negative. Urine: albumin, negative; sugar, negative; no red blood corpuscles; no casts. Spinal fluid: 21 cells to the c.mm.; not under pressure; Fehling's reduced; globulin, negative; Wassermann, negative.

Patient's Condition While in Hospital. He ran a normal temperature and his mental condition was clear. For the first week the masked expression became more marked, as did the tendency to stare: furthermore, he developed a partial ptosis of both lids.

The twitching of the right trapezius remained constant and was at the rate of sixty times a minute; a marked quivering of the muscles of the arms and tremors of the hands were noted. He responded to questions very slowly and articulated with great difficulty. He slept but little. He improved slowly but satisfactorily, but his relatives insisted on taking him home on the first of June, some time before it was advisable.

Patient's Condition One Year Later. There was a marked shakiness, but no disturbance in the region of neck or trapezius muscle. The tremor of the tongue was very noticeable. He complained of a continuous headache in the frontal region, had troubled sleep and was depressed about himself. He had worked part time for the past few months, but was under a doctor's care for two months after he left the hospital. He had gained considerable weight. The pupils were equal and reacted promptly to both light and accommodation. There was a slight ataxia, but no ankle clonus or Babinski. The patellar reflexes were exaggerated.

CASE VII.—V. S., an Italian, aged twenty-four years, was admitted to the Episcopal Hospital, May 11, 1920. His family history was negative. His personal history was that he had enjoyed good health until February, 1919, when he had a severe attack of bronchitis. He had never been well since then, had lost some weight and had had occasional nightsweats. He worked with lead and had had some gastrointestinal flareups for the past ten months.

His chief complaint on admission was nervousness, which had begun one week previously: he felt very shaky and was unable to sleep. He was irrational at times during the night, yelling loudly and talking incessantly. He belched a great deal and had a feeling of suffocation, but did not hiccough. He had diplopia for several days and also a great deal of difficulty starting the stream in urinating. He noticed a day or so before admission that there was a marked twitching of the abdominal muscles and a marked tremor of his hands.

Physical examination showed a masked facies. There was a slight jaundice. The eyes reacted normally to both light and accommodation; there was no ptosis. The tongue did not tremble. The heart, lungs and abdomen were negative. The patellar reflexes were exaggerated; the Babinski was negative; there was no ankle clonus.

The laboratory findings were: Urine: specific gravity, 1008;

albumin, negative; sugar, negative; no red blood corpuscles; no casts. Blood: white blood corpuscles, 6400; Widal, negative; Wassermann, negative.

Patient's Condition While in Hospital. His predominant symptoms remained unchanged for several days, and in addition he complained of pains in the upper abdomen. The jaundice slowly cleared up. The patient's temperature did not go above 100° F.; in fact, was above normal only about ten days. His mental attitude became poor and he was sure he would never be well. He was very restless, and as the masked expression disappeared its place was taken by an appearance of sadness. However, the diplopia cleared up and his general physical condition improved very satisfactorily. He left the hospital June 16, 1920, with instructions that he should continue under medical supervision.

Patient's Condition One Year Later. He was weak for at least five months after his discharge. He complained of frequent headaches in the frontal region and of indefinite pains in the abdomen and back, probably psychic. He felt unable to work and mentioned a continuous sorethroat, although there was no evidence of even the slightest congestion. His eyes reacted normally to both light and accommodation. The patellar reflexes were exaggerated: there was no Babinski and no ankle clonus. He had a slight ataxia.

CASE VIII.—M. D., aged forty-two years, was taken sick June 4, 1920. His family history was negative. In his personal history he stated that he had had heart trouble following an attack of inflammatory rheumatism four weeks previously. He was convalescent from the myocardial break when he became lethargic.

His present illness began with a feeling of malaise and lethargy: he also noticed diplopia. He complained of no body pain, but dropped into a prompt and continuous sleep as soon as he answered a question. Physical examination showed a rather anemic-looking individual with a masked facies. His eyes reacted normally to both light and accommodation. The tongue had no tremor. Lungs and abdomen were negative, the heart showed a slight hypertrophy to the left; the muscular tone was fair; there was a blowing systolic murmur at the mitral area, but no arrhythmias. Kernig's sign was suggestive; there was no ankle clonus; his patellar reflexes were exaggerated. Tache was present.

The laboratory findings were: Urine: specific gravity, 1021; albumin, negative; sugar, negative; no casts; no red blood corpuscles; occasional leukocyte; amorphous urates. Blood: hemo-

globin, 80 per cent; red blood corpuscles, 4,100,000; white blood corpuscles, 9,600. Wassermann, negative. Blood-pressure averaged 120 systolic, 80 diastolic.

Notes on the Patient's Condition. His sleep became deeper and the second day after the illness began he was irrational at night; he was aroused with little difficulty, however, and answered questions intelligently. He complained of headache and his bowels moved only by means of an enema. At the same time he was unable to urinate, requiring catheterization every ten to twelve hours. He took liquid nourishment well and when aroused endeavored to coöperate in every way. He seemed to be doing well until the morning of June 12 when he showed evidence of acute cardiac and respiratory failure from which he died shortly after midnight the following day. His temperature varied between 99.2° and 101° until just before his death it reached 103.4° .

CASE IX.—W. H. R., aged twenty-four years, was admitted to the Episcopal Hospital on June 30, 1920. His family history was negative. In his personal history he stated that his health had been fair; he had had swamp fever at Paris Island while in the service, and had also had gonorrhea. His present illness began two weeks previous to admission with pain in the head, which followed a visit to the dentist and was accompanied by vomiting. The headache was almost constant, as was also the nausea, though he vomited only occasionally. At times he had been delirious. He had had difficulty in his speech and at times was unable to recognize his friends and did not realize what was going on around him. He was very sleepy most of the time, and seemed hazy about what had happened during his illness. His bowels were regular.

Physical examination showed a well-nourished man who was both conscious and rational. His eyes reacted normally to both light and accommodation; tongue had no tremor; heart, lungs and abdomen were negative. The patellar reflexes were exaggerated; the Babinski was negative: there was no ankle clonus.

The laboratory findings were: Urine: specific gravity, 1009; albumin, present; sugar, negative; few red blood corpuscles; few white blood corpuscles; few hyaline casts. Blood: hemoglobin, 95 per cent; red blood corpuscles, 4,510,000; white blood corpuscles, 10,600. Differential polynuclears, 74; lymphocytes, 26; Wassermann, negative. Spinal fluid: under pressure; smear of it was negative. Diplococci were found in smears from the nasopharynx. Culture from the nose and throat showed only *Staphylococcus albus*.

Patient's Condition While in Hospital. Patient remained in a deep lethargy continuously. In a few days, however, a suggestive Kernig's was obtained, as was also a tache. An eye examination revealed a swelling of both the right and left optic nerves. In spite of strong supportive treatment there was no improvement in his condition; the lumbar puncture, as a therapeutic measure, resulted in lessening his restlessness; it was done five times. His condition gradually became worse and he died on July 21, 1920, from cardiac and respiratory failure.

Our mortality is high, yet little more than Netter reports in Italy, where there were 1013 deaths out of 4000 cases. As a matter of fact, our percentage would have been much better had we included one case seen early in 1919 and another in February, 1920, in consultation out of town. Both were undoubtedly atypical cases and recovered.

DIFFERENTIAL DIAGNOSIS. In considering the differential diagnosis of lethargic encephalitis we must consider, (1) syphilitic meningitis, where the positive findings are fluid under pressure, positive Wassermann, positive Kernig's sign, some rigidity of the muscles of the neck and a patient in a continuous stupor and who when roused cannot comprehend what is said to him; (2) tuberculous meningitis, where there is also fluid under pressure and it contains many lymphocytes and tubercle bacilli, positive Kernig's, some rigidity of the muscles of the neck, the tuberculous history and physical findings and the dissociation between the temperature and pulse; (3) anterior poliomyelitis, in which the spinal fluid is cloudy, contains many polymorphonuclear and eosinophilic leukocytes and the meningococcus, positive Kernig's stiffness of the muscles of the neck, exaggerated patellar reflexes, positive Babinski and ankle clonus; (4) cerebral abscess, in which the eye examination showed choked disk, the leukocyte count is high, there is a marked pleocyte count and at times cloudy spinal fluid, hectic temperature, usual history of primary focus and projectile vomiting; (5) brain tumor, in which there is usually a history of slow onset with headache and vomiting.

TREATMENT. The treatment of lethargic encephalitis is entirely symptomatic, as must necessarily be the case in the present state of our knowledge. Absolute quiet, light nourish-

ing food given at frequent intervals, good nursing and hydrotherapy are fundamentals. Lumbar puncture as a therapeutic measure in cases where the meningeal symptoms are marked is very effective. Urotropin, in 10-grain doses three times daily, has been advised, but the results do not appear to justify its use.

CONCLUSIONS. 1. The protean character of the symptoms of lethargic encephalitis makes the diagnosis in the atypical cases very difficult.

2. Laboratory and clinical findings make it evident that mild and abortive cases and carriers play an important part in its spread, especially during an epidemic.

3. The four most prominent symptoms are eye conditions, lethargy, fever and atypical spinal fluid.

4. The filterable virus has a selective action on the brain-stem.

5. Definite symptoms of cerebral disturbance are seen in patients one year after the disease.

In conclusion, I wish to express my gratitude to Dr. Arthur H. Hopkins and Dr. John Eiman for their assistance in the preparation of this paper.

REFERENCE

1. Boyd, William: *Annals of Medicine*, July, 1920.

EPILEPTIFORM CONVULSIONS IN ALCOHOLIC INTOXICATION; CASE REPORT*

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HISTORICAL. In 1813 Sutton¹ described the occurrence of epileptiform convulsions in an intoxicated individual. Such convulsions, referred to by some writers as "alcoholic epilepsy" and by the laity as "whisky fits," have been recognized by Huss² and other writers since his time as being associated with both acute and chronic alcoholism. Convulsions are especially commonly met with in countries where absinthe is taken, due to the convulsive action of the oil of wormwood contained in absinthe. In England and America, however, convulsions due to this cause are of rare occurrence, and I shall omit further consideration of this topic from our present discussion.

Of 88,271 admissions (see Table) to the Episcopal Hospital during the years 1911 to 1927 inclusive, 614 cases were admitted to the medical and neurological services or receiving ward with a primary diagnosis of alcoholism. Of this number, 18 (2.9 per cent of alcoholics admitted) had convulsions either immediately prior to or during their stay in the hospital. This figure may be compared to 27 (4.4 per cent) with delirium tremens and 6 (1 per cent) with alcoholic hallucinosis.

Of those with convulsions, the ages ranged from thirty-four to sixty, half of them being in their fifties. They were of various races and occupations; all were white and all but one were males. The number of convulsions varied from one to many. In the first sample of urine examined the majority had albumin and casts, but these abnormalities later disappeared in most of the cases where further urinalyses were made. In 3 cases where blood-urea-nitrogen determinations were made,

* Reprinted, with additions and alterations from *Medical Clinics of North America*, 1929, xii 113.

the figures obtained were 12, 12.6 and 23.8 mg. per 100 cc. of blood. Lumbar-puncture findings were recorded in 5 cases, and of these the cells per cubic millimeter numbered 81, 25, 10, 4 and 0. The cerebrospinal fluid was noted as under 18 mm. Hg. of pressure in one case, 12 mm. Hg. in another and "greatly increased rate of flow" in a third; the spinal-fluid

DATA RELATING TO ALCOHOLIC CASES ADMITTED FROM
1911 TO 1927 INCLUSIVE

Year.	Admissions to hospital.	Cases of alcoholism admitted. ¹		Alcoholics with convulsions.		Alcoholics with delirium tremens.		Alcoholic hallucinosi.	
	Number.	Num- ber.	Per cent.	Num- ber.	Per cent.	Num- ber.	Per cent.	Num- ber.	Per cent.
1911	4015	36	0.9	2	5.5	1	2.8	0	0
1912	4433	20	0.5	0	0	1	5.0	0	0
1913	5319	28	0.5	2	7.1	1	3.6	0	0
1914	5922	17	0.3	0	0	1	5.9	0	0
1915	4534	43	0.9	1	2.3	1	2.3	0	0
1916	5159	56	1.1	2	3.6	9	16.1	1	1.8
1917	4541	11	0.2	2	18.2	2	18.2	1	9.1
1918	4332	14	0.3	0	0	2	14.3	0	0
1919	4227	15	0.4	0	0	0	0	0	0
1920	5259	41	0.8	0	0	0	0	0	0
1921	5043	37	0.7	0	0	1	2.7	1	2.7
1922	5416	89	1.6	2	2.2	4	4.5	0	0
1923	5572	100	1.8	0	0	1	1.0	0	0
1924	5834	27	0.5	2	7.4	3	11.1	3	11.1
1925	5821	51	0.9	2	3.9	0	0	0	0
1926	6312	21	0.3	2	9.5	0	0	0	0
1927	6532	8	0.1	1	12.5	0	0	0	0
Total	88271	614	0.7	18	2.9	27	4.4	6	1.0

¹ Here are included only cases filed under alcoholism as the primary diagnosis. Inasmuch as extraneous factors enter into the admission and listing of these cases, the figures here given cannot be regarded as an index of the incidence of alcoholism in the community.

Wassermann and colloidal-gold reactions being negative. The average number of days spent in the hospital was six, the longest stay was thirty days, the shortest was several hours. No deaths occurred; the condition on discharge being recorded as "good" in 13, "fair" in 4 and "poor" in one, who left against advice. In addition to convulsions in 2 (11 per cent) of the group, the diagnosis of delirium tremens was

made and 4 others (22 per cent) were diagnosed as alcoholic hallucinosis. Our experience here is at variance with the usually accepted teaching that alcoholic convulsions usually appear as a complication of delirium tremens.

CASE REPORT; (unilateral alcoholic convulsions).

S. S., a white female aged fifty-two years, had a history of having been a heavy drinker for many years. In spite of this, except for attacks of headache and vertigo, her health was always good until ten years ago, at which time she had a "stroke," which paralyzed the entire right side of the body and affected her speech. She gradually improved so that after eight weeks she was able to be up and about, but she did not recover full strength on the paralyzed side of her body, and noticed, when fatigued, that the right foot had a tendency to drag a little. No complaints referable to her heart, lungs or kidneys could be elicited, but the attacks of vertigo and headache became so severe that she entered this hospital in 1923. Her clinical record at that time showed the blood-pressure to be 175 systolic and 105 diastolic, the Wassermann negative, the urine to have a specific gravity of 1.010, a trace of albumin and occasional hyaline and granular casts. The positive physical findings noted were moderate sclerosis of the radial arteries, ankle and patellar clonus on the right side and a dusky appearance of the optic disk. She was discharged and her condition remained unchanged until about two years ago, when she had an attack of convulsions about which little information could be elicited except that the movements may have been limited to the right side of the body and that the convulsions lasted about one day. She made an uneventful recovery from this attack and resumed her customary habits of living until December 20, 1927, when, after drinking more whisky than usual and while playing cards, she was suddenly seen to become tense and to stare at the ceiling apparently unable to speak or move. After some minutes she recovered consciousness and was put to bed. On the following morning she began to have convulsions and had already had seven when she reached the hospital.

Examination reveals a somewhat obese white female in a deep state of coma, which is interrupted at intervals by convulsions, during which she grinds her teeth, turns the head and eyes to the right, while the left side of the face, left arm and left leg undergo continuous clonic convulsive movements, the rest of the body including the abdominal muscles being held in a state of rigidity.

These attacks last from one-half to two minutes, and eighteen have been recorded in the space of one and a half hours. After each convulsion the patellar biceps and triceps reflexes are absent for a few minutes and then gradually return. Between convulsions the left arm and left leg are flaccid, while the right arm and right leg are spastic. The right foot gives a questionable Babinski sign. The blood-pressure is 150 systolic and 90 diastolic; otherwise nothing worthy of note is found on physical examination. The temperature on admission was 101° F. by axilla; later 104° F. Her leukocytes number 14,000, of which 90 per cent are polymorphonuclear cells. The fasting sugar, urea nitrogen and uric acid of the blood are 134, 23.8 and 5.7 mg. per 100 cc., respectively. The urine has a specific gravity of 1.015, contains a light cloud of albumin and a few hyaline and light granular casts, but is otherwise negative. The report on the spinal fluid shows that it came out under 12 mm. Hg. pressure, contained 4 cells per cubic millimeter, did not reduce Benedict's solution, was normal as regards globulin content and gave a negative Wassermann and colloidal-gold reaction. The Wassermann reaction of the blood is also negative. An x-ray of the skull is negative for fracture.

DISCUSSION. Whether the story of a previous attack of unilateral convulsions involving the opposite side of the body from the present attack, is to be believed is open to doubt. The nature of the "paralytic attack" which occurred ten years ago, involving the right side of the body, is also open to question. In view of the history of chronic alcoholism with unusual overindulgence for the past few days, alcoholic convulsions would appear to be a plausible diagnosis, although unilateral convulsions due to this cause are somewhat unusual. Jacksonian epilepsy due to subdural hemorrhage or cerebral neoplasm must, of course, be borne in mind as an alternative diagnosis.* Even if the history of this case were not known, the fact that an individual between the ages of thirty-five and fifty-five begins to have convulsions, should make us suspect alcoholism, especially if syphilis can be ruled out, as is the case here. Alcoholism and syphilis are the two most common causes for convulsions appearing in middle life.

* The later history of this case (cessation of convulsions and return to consciousness on the second day after admission, with disappearance of all abnormal physical signs and rapid and complete recovery) bears out the diagnosis of alcoholic convulsions.

The presence of fever by no means speaks against the diagnosis of alcoholic convulsions. The Episcopal Hospital records of 435 cases of alcoholism, in which temperatures were recorded, show that 92 (21.1 per cent) had temperatures over 99.6° F., the highest being 104.2° F. In the great majority of these cases no complications were noted. The presence of a leukocytosis, likewise, cannot be regarded as evidence against an alcoholic etiology in this case. Of 104 cases of alcoholism whose records contain a leukocyte count, 27 (26 per cent) exhibited leukocytoses ranging from 11,000 to 23,600. Leukocytosis and fever did not appear to be correlated either with each other or with the presence of gastritis, bronchitis or infection elsewhere. They were, however, more common in the cases with convulsions than where uncomplicated alcoholism occurred: of 8 cases with convulsions in which leukocyte counts were recorded, 5 (62.5 per cent) exhibited leukocytoses of 11,000 or over, and of 15 in which the temperature was recorded, 12 (80 per cent) had fever of 99.6° F. or over. These figures, it will be seen, are more than double those for the alcoholic group as a whole. In this connection it is of interest to note that leukocytosis and fever may also be present in idiopathic epilepsy.³

Epileptiform seizures occurring during alcoholic intoxication are regarded by some writers as due to epilepsy which has remained latent until unmasked by the effects of alcohol. Dandy and Elman⁴ have succeeded in producing a somewhat analogous condition in experimental animals by unilateral injury to the motor cortex. After a lapse of time sufficient for fibrous-tissue formation, they were able to produce unilateral convulsions, corresponding in location to the damaged cortical area, by the administration of absinthe in dosage far below that required to produce convulsions in normal animals.

Another theory which might explain the convulsions seen in alcoholics is suggested by MacNider's⁵ observations upon naturally nephropathic dogs. He found that the administration of alcohol to such animals resulted in a marked increase in the albumin and casts of the urine, a diminution in the excretion of phenolsulphonephthalein, an increase in the blood-urea nitrogen and sometimes a temporary anuria. It is con-

ceivable, therefore, that a person with diminished renal function might be thrown into a state of uremia through the agency of alcohol with resultant uremic convulsions. The case presented was known to have had hypertension and nephritis four years before her present admission. The present urine and blood studies do not, however, warrant the diagnosis of uremia. The urines of this patient and 3 others suffering from alcoholism with convulsions have been examined since their discharge from the hospital. The results did not indicate the presence of nephritis.

There is yet a third hypothesis to explain alcoholic convulsions; it is suggested by the experiments of Elsberg and Pike.⁶ They found that in cats to whom small doses of absinthe had been administered an elevation of the intracerebral pressure (which they produced by the intravenous administration of hypotonic solutions) was accompanied by convulsions. On the other hand, to produce convulsions in animals to whom hypertonic solutions had been administered, with a resultant *diminution* of the intracerebral pressure, doses of absinthe in excess of those producing convulsions in normal animals had to be administered.

TREATMENT. Whatever may ultimately prove to be the mechanism producing alcoholic convulsions, it would appear that they are most effectively treated by measures which are directed to dehydration and the lessening of intracerebral pressure. Frequent lumbar punctures, sharp restriction of fluids, saline cathartics by mouth and 50 per cent glucose solution intravenously are the methods employed.

PROGNOSIS. The prognosis in alcoholic convulsions, in our experience, is by no means as bad as has been generally believed. Out of 18 cases, so diagnosed in this hospital in the past seventeen years, none have died, and the majority were discharged in good condition in less than a week after their admission.

SUMMARY. 1. Alcoholic convulsions may occur in cases of either chronic or acute alcoholism.

2. In any case where the cause of convulsions appearing in middle life is being sought, alcohol should be considered.

3. Leukocytosis and fever often accompany alcoholic intoxi-

cation, but are respectively about two and one-half and four times as common in alcoholic convulsions as in alcoholism without convulsions.

4. Delirium tremens was absent in the majority of our cases of alcoholic convulsions, and recovery was generally rapid, not a single death from this cause occurring in the group studied.

5. Increased intracerebral pressure appears to be a factor in the production of these convulsive seizures, and measures directed toward lessening such pressure are recommended.

6. Unilateral convulsions due to alcoholism may occur. A case so diagnosed is presented.

BIBLIOGRAPHY

1. Sutton, T.: *Delirium Tremens and Gout*, London, 1813, p. 38, footnote.
2. Huss, M.: *Alcoholismus Chronicus*, Stockholm and Leipzig, 1852, pp. 314 and 316.
3. Muskens, L. J. J.: *Epilepsy*, London, 1928, p. 259.
4. Dandy, W. E., and Elman, R.: *Bull. Johns Hopkins Hosp.*, 1925, xxxvi, 40.
5. MacNider, W. D.: *Jour. Pharmacol. and Exp. Ther.*, 1925, xxvi, 97.
6. Elsberg, C. A., and Pike, F. H.: *Am. Jour. Physiol.*, 1926, lxxvi, 593.

EDEMATOUS ANTHRAX OF THE FACE RESULTING IN MENINGITIS¹

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IN this case there was no local skin lesion and an incision of the lid revealed no bacilli. This made the case very obscure. The anthrax bacillus was discovered only after spinal puncture was made. The general features of such infections are reviewed in contrast to the symptoms of the case.

P. M., aged thirty-five years, a piecer of yarns, presented himself at the Eye Dispensary of the Hospital of the Protestant Episcopal Church with the history of having had a small swelling, thought to be a sty, on the upper lid of the right eye two days before. He had no pain, but consulted his family physician, who prescribed for him on account of the swelling of his face. The swelling increased enormously in spite of the treatment.

When seen at the clinic, there was an intense swelling of both lids of the right eye, extending over to near the parotid region. There was no external lesion of any kind to account for the edema, nor could any area of greater density be localized by sense of touch. A small separation of lids, made forcibly with a retractor, showed the cornea and iris to be normal; but no eversion of the lids was possible, nor was there any bloody discharge from the use of the retractor.

A deep incision in the upper lid, made by my associate, Dr. Brinkerhoff, resulted only in a bloody discharge. The culture made was negative. A hot saturated solution of magnesium sulphate was ordered for continuous application, the patient to

¹ Reprinted from the American Journal of Ophthalmology, 1926, ix, 337.

return the following day if not relieved. He returned two days later to the clinic of a colleague, Dr. A. G. Fewell, where the patient had a convulsion.

Dr. Fewell, recognizing the seriousness of the affection, admitted the patient into the wards and notified us. When we saw the patient that night, he had a temperature of 102° . The swelling had increased so that the whole of the right face was involved. The swelling was hard, brawny, with a rather sharply defined edge as in erysipelas. The upper lid at the site of the wound was purplish, the edges of the wound somewhat necrotic. Culture from this wound and from fresh incisions showed a few cocci.

The patient was seen by various members of the staff of the hospital, but, as the lesions were masked, anthrax was not suspected. The *x*-ray of the sinuses was negative. The urinary analysis and Wassermann test were negative. The white blood count was 10,800; the blood culture was negative. The neck, chest, abdomen and extremities were negative. The teeth were reported to be in bad condition.

The day following his admission to the house the swelling, while extensive, seemed somewhat less on the right side, but was encroaching the left side over the bridge of the nose. That evening he vomited and later became very restless. He was quieted for a few hours with a morphine injection given by Dr. Spikes; when he awoke he was delirious. He showed some rigidity of the neck but had no Kernig's sign. The temperature remained at 102° . He became comatose, with Cheyne-Stokes respiration. The neck became spastic, hand and fingers athetoid. Spinal fluid was withdrawn, the examination of which showed a fluid that was very cloudy, with sugar present, a globulin cloud and a cell count that was impossible on account of much coagulated albumin.

The smear of the fluid showed very many pus cells and anthrax bacilli. The growth showed pure culture of anthrax bacilli. Pulmonary edema developed rapidly, the patient dying at 3 A.M. the following morning, within thirty-six hours of his admission to the hospital.

The autopsy showed a suppurative meningitis due to anthrax bacilli, which were found both in smear and culture. A culture from the right eye made at the postmortem showed no anthrax bacilli. The postmortem changes consisted of extensive edema and hemorrhages in all of the deeper structures. In the stomach it was in the submucous coat. In the lungs, edema and congestion were

present with hemorrhages into their deeper structures. The meninges were congested, with extensive hemorrhages in the subarachnoid space.

In this patient we had a mysterious swelling of the right side, with no local lesion and no evidence of sinus disease. There was a marked absence of pain. A diagnosis of intense cellulitis of unknown origin had been made. This was amplified just before death when anthrax bacilli were found in the spinal fluid, showing that we had a case of malignant edema. The subject of anthrax is one that does not often come to the attention of ophthalmologists. The case reported will show the rare importance of remembering that anthrax can assume an unusual appearance.

A short review of anthrax infection may not be out of place.

Anthrax is a disease caused by a specific organism, the anthrax bacillus, which is a rod-shaped, nonmotile organism remarkable for its large size, about $\frac{1}{3000}$ inch in length. When growing in living animals, it multiplies by fission and no spores are formed. If, however, the blood or any discharge from an infected animal be exposed to the air, small round spores of remarkable vitality are formed in the bacilli. These spores may survive for several years and remain capable of reproducing the disease in a favorable media. They resist antiseptics, but are killed by boiling for fifteen or twenty minutes.

The disease essentially is one that attacks cattle, sheep, horses and mice, carnivorous animals being much less susceptible. It is communicated to man usually by contact with living or dead animals or with the hides of the infected animals.

The disease begins usually as a cutaneous lesion, known as malignant pustule. When the respiratory tract is the primary seat of infection, it is known as woolsorter's disease. In Guy's Hospital, of 100 cases reported only three women were infected, the rest being largely waterside laborers, porters of raw hide (about 58 per cent) tanners and leatherdressers; 12 per cent of the rest being in various occupations. In this series the pustule was situated in the face of 48 patients and on the neck of 41.

Among the symptoms in the ordinary case of malignant

pustule is the formation of a pimple or pustule, usually thought to be the bite of an insect. The pain, redness and swelling increase, so that usually on the third day a physician is sought. The typical lesion, then, is a pustule one-quarter of an inch in diameter, in the center of which is a black slough due to extravasated blood. Surrounding this is a ring of vesicles filled with a clear or semipurulent fluid. Around the vesicle is a red indurated area, beyond which is edema of the skin and subcutaneous tissues.

In some cases the black slough or vesicles are absent, and in early cases the redness and edema may not be present. Occasionally there is no definite pustule, but a large brawny area with much edema, simulating an ordinary cellulitis. This condition is known as anthrax edema. When present in the neck there is serious danger of edema of the glottis. Constitutional symptoms vary in severity, but dizziness and headache are usually present. The temperature runs about 102° to 103° . In fatal cases a form of septicemia is produced.

Even when there is extensive edema, with thrombosis of the subcutaneous veins and several constitutional symptoms present, a considerable proportion of patients recover under active treatment. This should consist in excising the pustule, the incision extending to the deep fascia. The raw surface is then treated with pure carbolic acid, sulphur emulsion or actual cautery. The wound is allowed to granulate. If there is much edema, a 20 per cent carbolic solution is injected about the excised area. Anthrax serum is found to be of much value, but early excision should always be the primary treatment. Finally, meningitis is not a common feature in this disease. In our case it directly gave us our diagnosis, as before the spinal puncture, due to the development of cerebral symptoms, we knew no causative factor.

F. Herzog¹ quotes 3 cases in which there was a hemorrhagic leptomeningitis, with extensive hemorrhage in the subarachnoid and pial layers of the brain, due to degeneration or solution of the arterial wall, the outer layers of which seem to be

¹ Beit. z. path. Anat. u. z. allg. Path. vol. lx.

particularly susceptible to the anthrax poison. Even in the lymphatic structures of the neck, similar hemorrhages were found, which destroyed the parenchyma of the gland.

Herzog quotes a number of German authors in his bibliography who have reported meningo-encephalitis in fatal anthrax, which in every case was of a hemorrhagic type. The blood-vessels were always widely dilated, with extensive exudates of red blood cells, large numbers of lymphocytes, much altered by the intensity of the pathological irritation. Some of these cells contained large vacuoles and were not apparently cytophagic in function.

PARTIAL CONTINUOUS EPILEPSY

WITH ESPECIAL REFERENCE TO THAT PRODUCED BY
MICROSCOPIC CORTICAL LESIONS*

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KOJEWNIKOFF,¹ Orłowski² and Choroschko³ have described cases of epilepsy in which, during intervals between the general attacks, continuous muscular twitchings appeared at different places while consciousness was not affected. To this condition Kojewnikoff gave the name of *epilepsia partialis continua*. He said that the cause of such a condition might be tumor, abscess, syphilis, edema, embolism or a localized encephalitis. In his case he concluded it was localized encephalitis with sclerosis. This conclusion was reached without necropsy findings. Since the original description by Kojewnikoff and the articles mentioned above, partial continuous epilepsy has been discussed by Spiller,⁴ Burr⁵ and others. Spielmeyer⁶ and Mills⁷ have reported cases in which, from their descriptions, partial continuous epilepsy may have been present, although the cases were cited under the title of Jacksonian epilepsy.

Spielmeyer reported instances of Jacksonian epilepsy arising in cases of encephalitis in the motor cortex where there was perivascular infiltration mainly of the plasma cell type. Spielmeyer's case was similar in many respects to our Case II. In Mills' case a circumscribed hemorrhagic cortical encephalitis limited to the middle third of the motor zone was present.

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Microscopic examination showed that the minute hemorrhages were almost all confined to the cortex, although a few were present in the white matter. The cause of the partial continuous epilepsy in Spiller's case was meningoencephalitis of syphilitic origin, and Burr's patient had a secondary carcinoma of the cortex.

Patients with partial continuous epilepsy have an irritative lesion of part of the motor cortex; this is usually a gross lesion, but it may be microscopic. We wish to report three cases of the latter type.

The importance of this syndrome in a patient with focal encephalitis is obvious because operation is contraindicated, whereas in partial continuous epilepsy due to tumor operation is always in order.

CASE I.—*History.* A white girl, aged two years, was admitted to the Episcopal Hospital on November 21, 1922, and died on November 25, 1922. On September 7, 1922, the patient fell from a baby carriage, striking her head; she was unconscious for a few minutes. She bled profusely from the nose and from laceration of the forehead. She did not vomit, but was drowsy for twenty-four hours after the accident. For one week following the fall she showed a tendency to lie around rather than to play. On the eighth day it was noticed that the right leg gave way when walking. On the tenth day the right side of the face began to twitch, and on the fourteenth day the right hand became weak and the child was no longer able to use it in feeding herself. The paralysis progressed and the twitchings of the right side of the face persisted until she was admitted to the hospital about ten weeks after the fall.

Examination. The child lay on her back, apparently unconscious. The right extremities were spastic, with exaggeration of the deep reflexes, ankle clonus and a Babinski sign. The most striking feature of the child's condition was the rhythmic contractions of the right side of the face with extension of the fingers of the right hand and of the wrist. These movements were synchronous, occurred two or three times a minute, and kept up continually for the four days the child was in the hospital.

Examination of the eyes by Dr. Fewell revealed no abnormalities.

Blood examination revealed: red blood cells, 3,890,000; white blood cells, 10,400; hemoglobin, 70 per cent. Spinal fluid examination revealed: a slightly bloody fluid, 22 cells to the cubic milli-

meter, all lymphocytes, a negative globulin and a negative culture. The Wassermann test was negative.

A roentgenogram of the skull by Dr. Bromer showed no fracture.

The temperature was 102° F. on admission, the following day 101° F., and the last two days of her life 100° F.

Operation. Operation was performed by Dr. Ashhurst on November 25, 1922, exposing the lower part of the left motor cortex. The dura was adherent and thickened over an area 1.5 cm. square in the lower part of the exposed area. Dr. Ashhurst was of the opinion that there was an area of meningitis along the sylvian fissure, and a culture was made from the meninges, the result of which was negative.

Pathologic Study of Brain. No gross abnormalities were noted. Sections were made from various parts of the motor zones and representative areas from the rest of the cortex, from the basal ganglia, midbrain, pons and medulla, stained with hematoxylin-eosin, phosphotungstic acid—hematoxylin, sharlach R., toluidin blue and Weigert's myelin sheath stain.

The piaarachnoid was somewhat thickened owing to edema, marked congestion and a slight infiltration of lymphocytes with a few gitter cells. The vessel walls were normal.

The cortex was definitely altered, more in the left motor area than elsewhere. Here there were focal accumulations of cells (Fig. 1), lymphocytes and the polyblasts of Maximov (Fig. 2). The ganglion cells in the neighborhood of these cell accumulations were either invaded by them (neuronophagia) or showed little change besides a simple chromatolysis. Both of these conditions occurred within the same focus. The same kind of cell accumulation occurred about some of the vessels, especially the veins (Fig. 3), but here an admixture with gitter cells was seen. All the vessels stood out because of marked congestion. These foci were present in every section examined. The basal ganglia, especially the left, showed many of the focal accumulations of wandering cells, much less in number than in the motor area, but greater than in some of the other parts of the cortex. These foci were for the most part limited to the gray matter. No such foci were found in the pons or medulla but an occasional vein collared with lymphocytes was seen in these parts. Sections stained for organisms were negative.

Pathologically, this case resembles to a slight degree the fulminating type of encephalitis described by Cleland and Campbell⁸ as the "X" disease in the epidemic of 1917-1918 in New South Wales; the process, however, was much less intense in our case.



FIG. 1.—Cortex showing two areas of focal infiltration at A. $\times 92$.

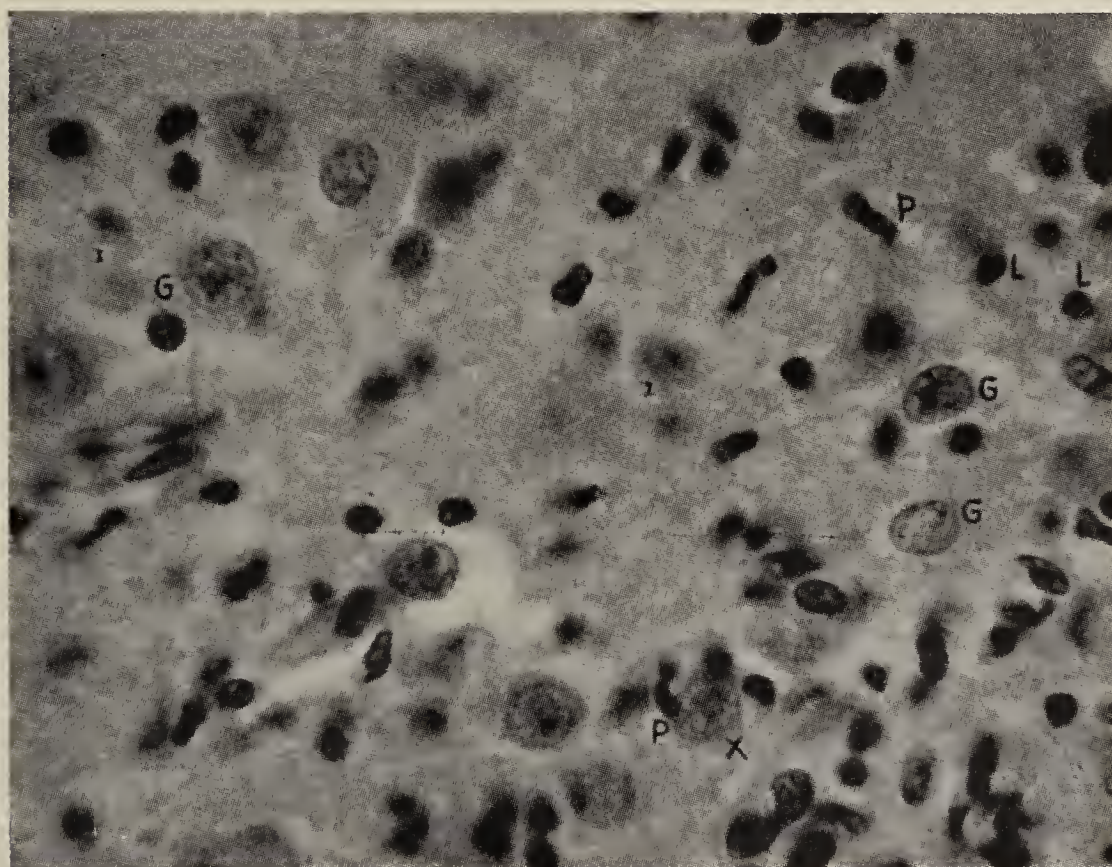


FIG. 2.—Focus A in Fig. 1 under oil ($\times 1000$) showing character of cells. G, ganglion cells (cytoplasm indistinctly shown); P, polyblasts; L, lymphocytes. At X there is a ganglion cell showing neuronophagia.

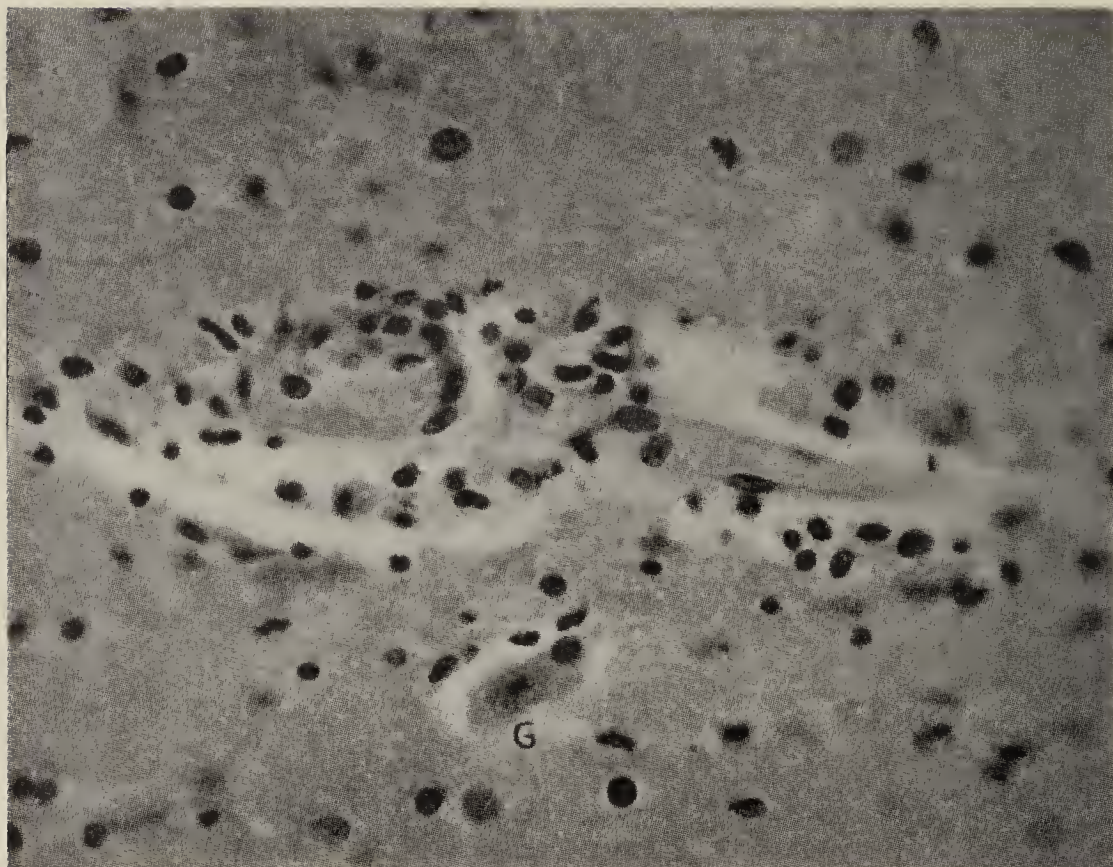


FIG. 3.—Focus similar to *A* in Fig. 1. The infiltration is mainly but not entirely perivascular. Involvement of a ganglion cell (neuronophagia) is shown at *G*. $\times 550$.



FIG. 4.—Showing marked infiltration of pia, mainly perivascular. $\times 115$.

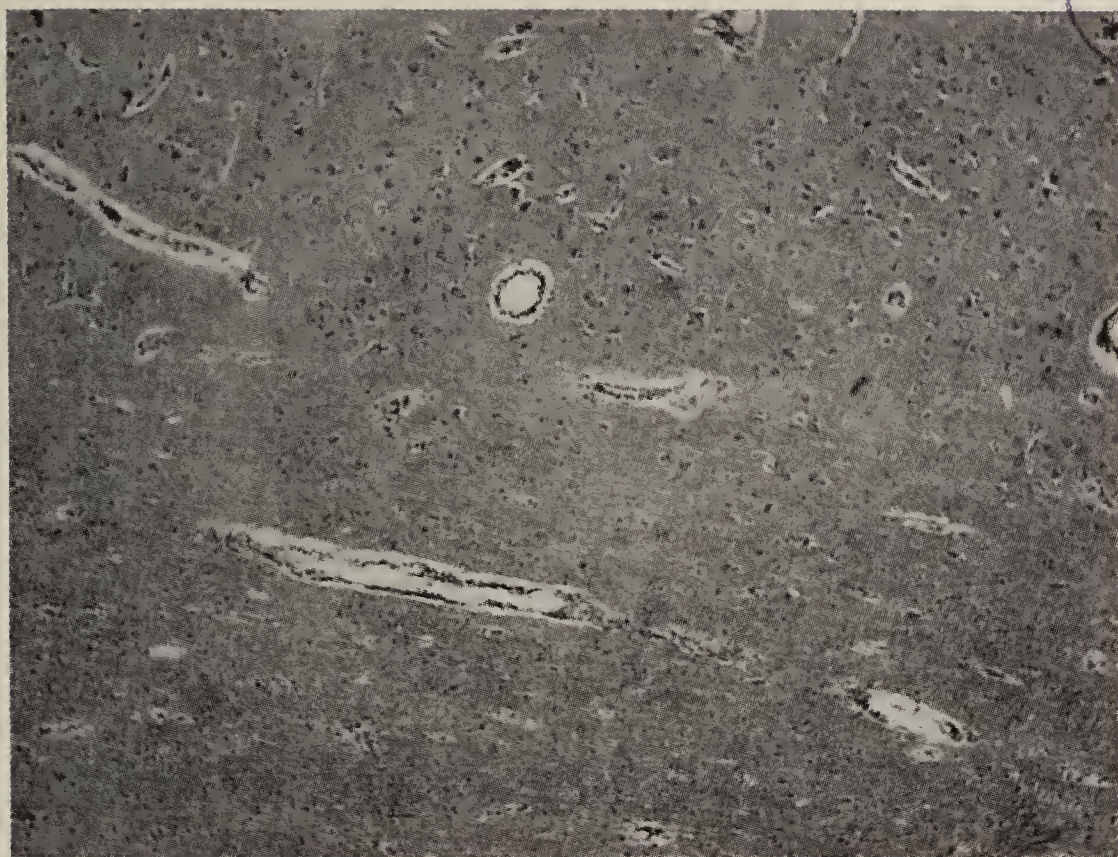


FIG. 5.—Vessels in cortex showing generalized collaring with lymphocytes and plasma cells. $\times 115$.



FIG. 6.—Area from basal ganglia showing one vessel with marked perivascular infiltration. $\times 184$.

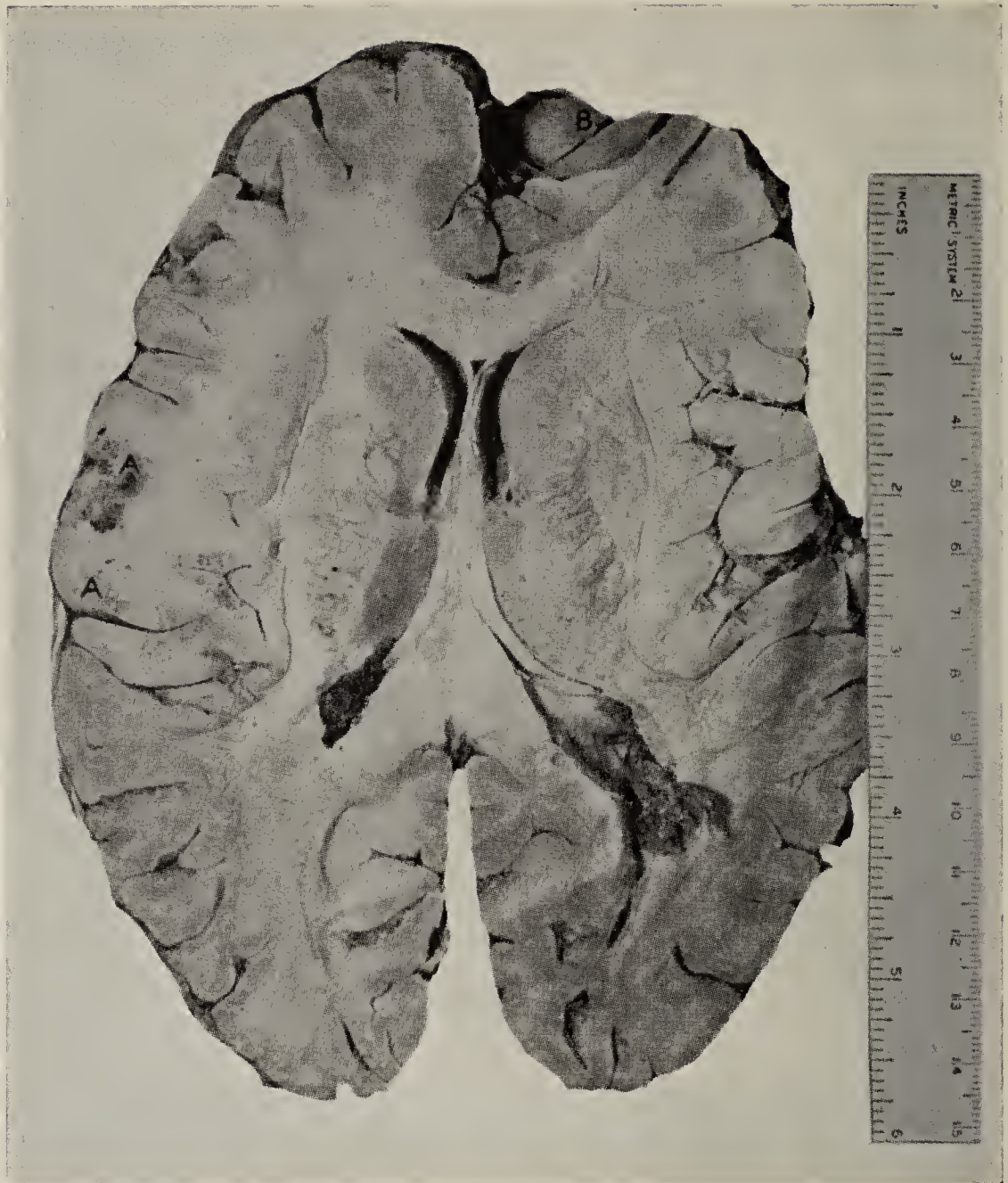


FIG. 7.—Transverse section of brain showing petechial hemorrhages at *A*.
A similar area occurred at *B* which was removed for microscopic study.

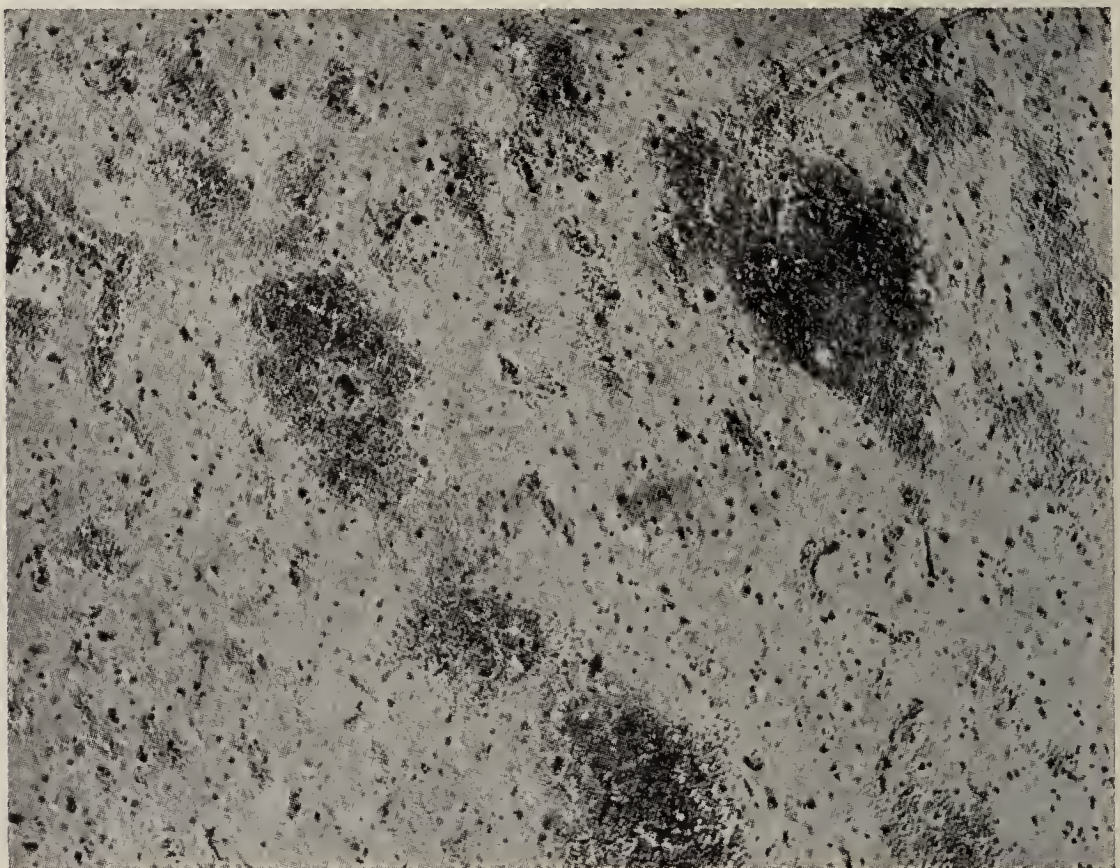


FIG. 8.—Petechial hemorrhages in left motor area.

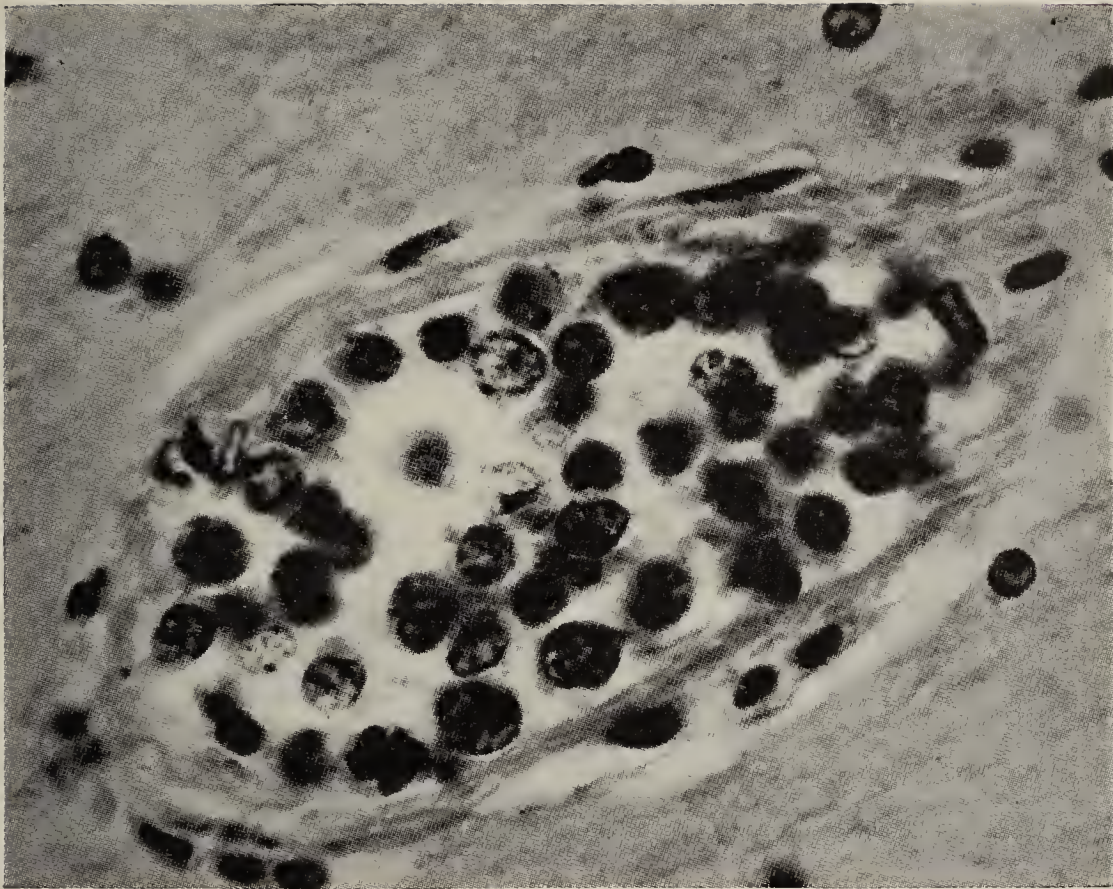


FIG. 9.—A vessel in the cerebral cortex almost completely filled with carcinoma cells. Blood elements while present are inconspicuous. $\times 1000$.

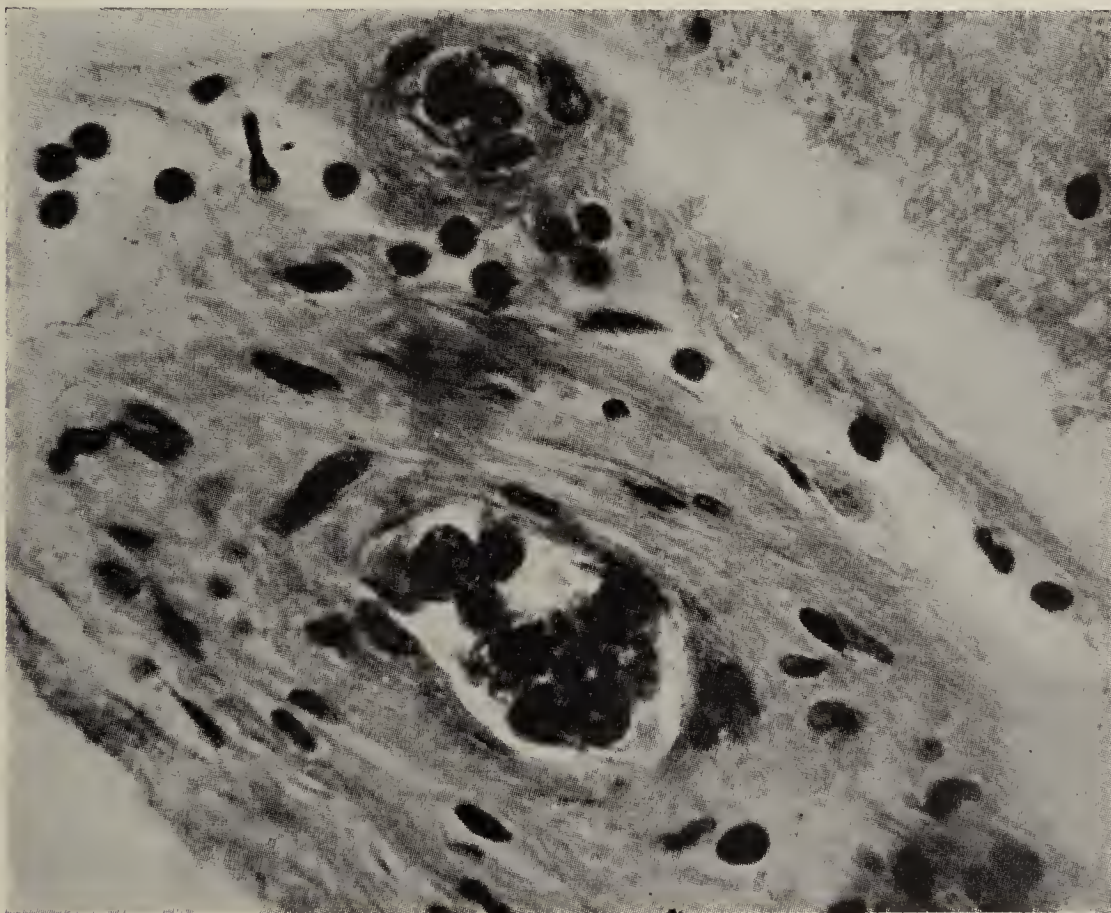


FIG. 10.—Two cortical vessels, both filled with cancer cells.

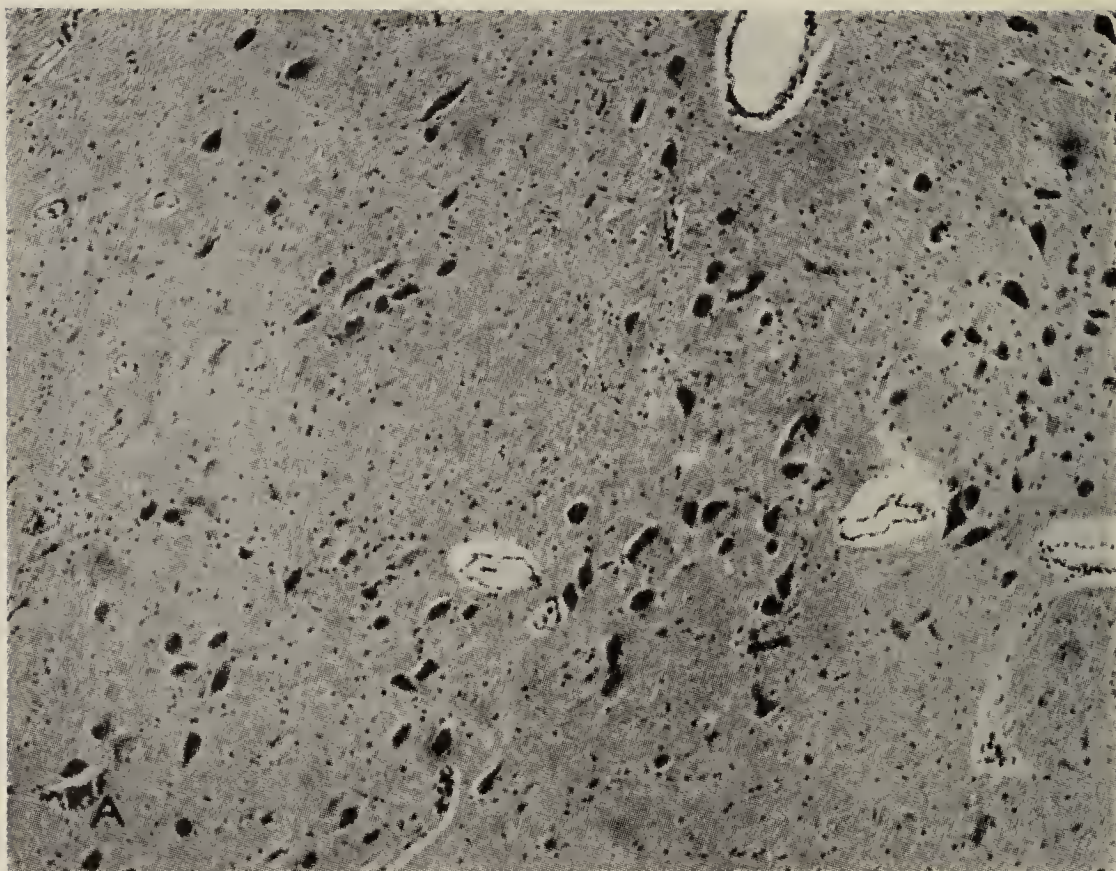


FIG. 11.—Low power magnification of substantia nigra showing cancer cells at A; other vessels show mild perivascular infiltration. $\times 115$.

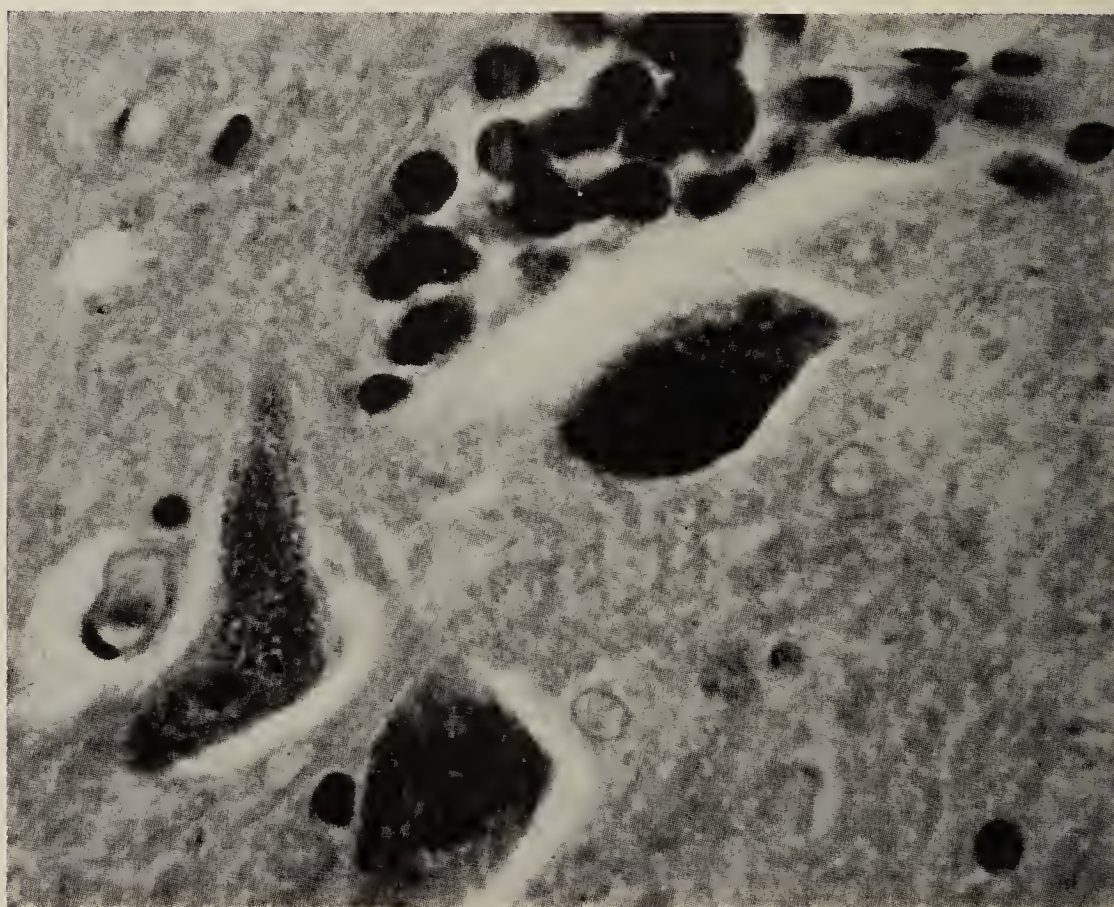


FIG. 12.—Area A from Fig. 11 under high magnification showing the vessel filled with carcinoma cells and three normal substantia nigra cells.

It also resembles the cases described by Meleney⁹ as encephalomyelitis. The type of cell described in both these papers, and called by the authors of the former "lymphocyte-like" and by those of the latter "large mononuclear phagocytic type," resembles those found in poliomyelitis. We believe these cells are derived from lymphocytes and are comparable to the cells designated by Maximov as polyblasts. That the lesions were practically limited to the gray matter is significant. The pathologic diagnosis is encephalitis, cortical type, unknown etiology.

Comment. The history and the onset of symptoms immediately after the accident led to a diagnosis of subdural hemorrhage, hence the operation was performed. Certain features in the case pointed away from a traumatic affection, namely, fever, leukocytosis and the presence in the spinal fluid of twenty-two lymphocytes. The diagnosis of subdural hemorrhage, while incorrect, seemed to be justified. From a pathologic point of view the case is most unusual and resembled in many ways polioencephalitis.

CASE II.—*History.* A white, married woman, aged twenty-six years, was admitted to the University Hospital on April 13, 1916.

Five weeks before her admission to the hospital she experienced a feeling of numbness in the left lower extremity, especially in the thigh, with a feeling of weakness in the left leg. One week after the onset, while stepping from an automobile, the symptoms were suddenly aggravated, and she had a convulsion beginning in the left leg, becoming general, with loss of consciousness and biting of the tongue. She had six attacks. In addition to these major attacks, she periodically had special others which persisted for some time and in which the left hand and arm were the seat of rhythmical twitching. During the few days before her admission the partial continuous epilepsy in the left arm was marked and persisted practically all the time.

The patient had been married for six years, had one living child, aged five years, and had had three miscarriages, all occurring at about the third month.

Examination. All the deep reflexes were present and exaggerated. The plantar reflex on the right was normal, on the left there was no response. The grip was good on both sides, and the movements of the upper extremities were strong. The left upper extremity showed impairment of the sense of position with distinct astereogno-

sis in the left hand. The left upper extremity was the seat of continuous twitching, most marked in the fingers and at the wrist, consisting chiefly of extension and flexion of the wrist and separation of the fingers.

In the left foot, there was a movement of inversion and eversion at intervals of from eight to thirty seconds. Mentally, the woman was confused and emotionally unstable.

Results of examination of the eyegrounds, blood, urine and spinal fluid were negative.

Operation and Course. Because of the distinct focal features it was thought that the patient had a brain tumor, and an exploratory craniotomy was performed by Dr. Charles H. Frazier. The flap was made over the right rolandic fissure. On reflection of the dura, there was seen at the upper margin of the opening, more behind than in front of the rolandic fissure, a confluence of veins which seemed to converge toward the leg center and which were especially marked over the foot center. One of these veins was as large as a slate-pencil. At this time no further exploration was made except palpation of the cortex, in which a number of adhesions were found. This part of the brain was softer than normal, but no positive evidence of either a cortical or subcortical growth was found. The patient died two days after the operation.

Microscopic Examination. Only the right half of the brain was obtained. Grossly, we could discover little because of the length of time the brain had been in liquor formaldehydi (seven years).

Sections were taken from all representative cortical areas and also the basal ganglia, pons and medulla. These sections were stained with hematoxylineosin, phosphotungstic acid—hematoxylin, sharlach R., toluidin blue and Weigert's myelin sheath stain.

The piaarachnoid varied over different areas. Over the occipital lobe there was quite marked infiltration with round cells, mainly lymphocytes; also a few plasma cells and many gutter cells (Fig. 4). These cells were particularly noticeable in the vicinity of vessels, especially in the perivascular spaces. Over the motor and parietal areas many large veins filled with degenerating blood were seen, while the cell infiltration was scanty. The bloodvessels in the pia showed little or no thickening; an occasional swollen endothelial cell was seen.

The cortex varied in different localities. In the occipital lobe particularly, although present also in the motor, parietal, frontal and hippocampal areas and in the basal ganglia, many vessels (especially veins) were seen to be "collared" with a mass of round

cells in their perivascular spaces—cells that were of the same nature as those seen in the piaärachnoid, and they occurred in both gray and white matter (Fig. 5). The vessel walls showed no marked change, especially none that might be interpreted as syphilitic. No new vessel formation could be made out. There was no gross alteration in the cytoarchitecture, but many cells showed neurophagia. A few rod cells were present.

In the motor and parietal areas, recent petechial hemorrhages were noted in both gray and white matter, while in the same neighborhood beginning abscess formation was seen with collections of polymorphonuclear cells in the piaärachnoid over the immediate location. An excess of glia cells was not evident.

The basal ganglia sections showed the same vessel changes (perivascular infiltration) but to a much less degree (Fig. 6). The pons and medulla were normal.

Pathologic diagnosis: Encephalitis not syphilitic but of unknown etiology.

Comment. The symptoms in this case pointed clearly to a motor parietal lesion, and because of the presence of partial continuous epilepsy on the left side the diagnosis of tumor was made. The most striking thing discovered at the operation was a tremendous enlargement of the veins on the upper part of the motor cortex, although the enlargement of the veins also was seen over the parietal lobe. Microscopically, encephalitis was found.

CASE III.—*History.* The patient, a white woman, aged fifty-four years, was brought to the Philadelphia General Hospital on May 4, 1923, by the police, who found her wandering on the streets. The woman was much confused and was unable to give a coherent account of herself. Her brother said that she was working as a shirtmaker on the day she was taken to the hospital. He said that she had not been well for about three weeks previous to admission. During this time she had complained of double vision; her speech was thick, and she had weakness of one side of the face. She had had both breasts removed, the first in February, 1918. Microscopic examination showed a scirrhus carcinoma. The other was removed a few years later, but a report on the pathology was not available.

Examination. The woman was emaciated, disoriented and confused. The scars of her operations were completely healed,

and there were no local recurrences, although enlarged glands were felt along the carotid vessels and in the supraclavicular spaces. In walking she always staggered to the left. The left pupil was larger; both reacted sluggishly to light. The deep reflexes were normal. The plantar reflex was extensor on the left and indifferent on the right. The lower extremities were hypersensitive to pressure.

Course of Illness. During the eight days she lived after admission to the hospital she had a series of generalized convulsions, in addition to which she had twitchings confined to the right side of the face. The eyegrounds were normal. Bladder and rectal control was lost. Spinal puncture showed no increase of pressure, but the fluid contained blood. Chemical study of the blood showed urea 43; uric acid, 6.8. Urinalysis showed a slight trace of albumin.

The woman gradually became more stuporous, and she died eight days after admission. The twitchings of the right side of the face continued until shortly before death.

Pathologic Study of the Brain. Grossly, the brain showed petechial hemorrhages in the face and arm centers of the motor cortex on the left side, but the hemorrhages extended into the parietal lobe and were entirely within the gray matter (Fig. 7). A similar hemorrhagic area was present in the right frontal pole.

Microscopically, the piaärrachnoid showed mild yet definite infiltration with lymphocytes which were chiefly perivascular in distribution. The vessels themselves showed a moderate degree of arteriosclerosis. No gutter cells were found in the pia or cortex.

Petechial hemorrhages were found not only as indicated before (Fig. 8), but also throughout the rest of the cortex, in the caudate nucleus and in the cerebellum, although to a much less degree, and in these parts not entirely confined to the gray matter. The bloodvessels, especially the smaller ones, contained cancer cells; some vessels were even completely occluded by these cells (Figs. 9 and 10). This condition of the vessels was present throughout the entire brain. In the basal ganglia, midbrain, pons and medulla vessels were seen "collared" by round cells, mainly of the lymphocytic type. The cells of the substantia nigra were uninvolved, but the vessels showed both perivascular infiltration and contained cancer cells (Figs. 11 and 12). In only one area was there found a perforation of the vessel wall by the carcinoma cells and their proliferation in the nervous tissue. This occurred in the left motor zone, the area most damaged by the petechial hemorrhages.

Metastatic nodules of considerable size were present in the lungs, and one rib was also involved. In the organs of the rest of the body no carcinoma cells were found in the vessels.

Comment. This case was one of generalized carcinomatosis with a superadded infection, although the round-cell infiltration attributed to the infection might have arisen as the result of the carcinomatosis. It is noteworthy that metastasis was taking place by way of the blood-stream. The petechial hemorrhages were in all probability due to blocking of the vessels by plugs of cancer cells and to local changes in the vessel walls.

The pathologic diagnosis was encephalitis due to carcinomatosis, with the possibility of an added infectious process.

The diagnosis made by Dr. C. S. Potts in this case was metastatic carcinoma, although he strongly considered the possibility of encephalitis. From the pathologic point of view the case was one of encephalitis produced by cancer cells which clogged up a great many of the vessels of the brain and caused gross petechial hemorrhages in two areas; one of these areas was responsible for the peculiar type of epilepsy under discussion. This case represents a rapid diffusion of cancer cells throughout the vessels of the brain and is similar to the condition in rodents when a mouse carcinoma is massaged. F. C. Wood¹⁰ has shown that, if a mouse tumor is massaged, in a few days the lungs will be filled with small thrombi consisting of carcinoma cells.

SUMMARY. When partial continuous epilepsy occurs in a patient, the natural tendency of most diagnosticians is to visualize a more or less gross lesion of the brain, such as a tumor abscess, subdural or extradural hemorrhage. In two of our cases operation was performed because the diagnosis, while proved incorrect by operation and necropsy, seemed logical. Our cases point out clearly that partial continuous epilepsy may be due to microscopic lesions in the cortex. It is important to realize this possibility because pathologic study of two of our cases showed that a great many cells were unaffected, and the chances for recovery at least to some extent were good.

When a patient with encephalitis such as that described in Cases I and II recovers, he will frequently have epilepsy and perhaps Jacksonian attacks at some time. It is, of course, a well-known fact that perhaps the most common cause of Jacksonian epilepsy is idiopathic epilepsy, and we believe that it is in this type of case that the so-called inside Jacksonian fit often occurs.

We desire to thank Dr. A. P. C. Ashhurst for permission to report Case I; Dr. Charles K. Mills for the privilege of reporting Case II, and Dr. C. S. Potts for the privilege of reporting Case III.

BIBLIOGRAPHY.

1. Kojewnikoff: *Neurolog. Centralbl.*, 1895, xiv, 47.
2. Orlowski: *Neurlog. Centralbl.*, 1896, xv, 521.
3. Choroschko: *Neurolog. Centralbl.*, 1908, xxvii, 279.
4. Spiller, W. G., and Martin, E.: *Epilepsia Partialis Continua Occurring in Cerebral Syphilis*, *J. A. M. A.* (June 12), 1909, lii, 1921.
5. Burr: *Am. J. Med Sci.*, 1915, cxlix, 169.
6. Spielmeyer: *Centralbl. f. Nervenheilk.*, 1904, xxvii, 371.
7. Mills: *Rev. Neurol. and Psychiat.*, 1907, v, 89.
8. Cleland and Campbell: *Brit. M. J.*, 1919, i, 63.
9. Meleney, H. E.: *Fulminating Encephalomyelitis*, *Arch. Neurol. Psychiat.* 1923, x, 411.
10. Wood, F. C.: *Notes on Tumors*, 1920, p. 46.

CARCINOMA IN AN ABERRANT THYROID AT BASE OF TONGUE*

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AND

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ABERRANT thyroid tissue in the tongue is unusual. Carcinoma occurring in such an aberrant thyroid is almost unknown. Bérard and Dunet¹ state that there is not yet on record a proved case of cancer of a lingual thyroid. The following is the history and pathologic report of such a case:

A. C., a man, aged fifty-six years, admitted to the Episcopal Hospital, November 22, 1916, complained of a lump on the back of his tongue near the right lower molar teeth. This lump had been noticed for more than twenty years; first as a small, hard mass that very gradually grew larger. After about five years it began to give him pain. Thirteen years previous to his admission to the Episcopal Hospital (*i. e.*, in 1903), this mass had been removed at the Methodist Hospital, Philadelphia, by Dr. Walter Roberts, who reported to us that it was thought to have arisen in the lingual tonsil and that microscopic examination had shown it to be entirely benign. Unfortunately, neither the sections nor the detailed report of the examination had been preserved.

The patient stated that about three years after this operation he noticed the glands beneath the jaw increasing in size, first on the right side of the neck, later on the left side; the growth on the tongue gradually returned. He then came to the hospital on account of difficulty in eating and swallowing and pain in the tongue.

The mass was about 8 cm. long in its anteroposterior diameter, on the right side of the tongue involving its posterior half; its

* Reprinted from the Journal of the American Medical Association 1925, lxxxv, 1219.

¹ Bérard and Dunet: Cancer of the Thyroid, Paris, 1924, p. 454.

posterior border, close to the epiglottis, was raised 2.5 cm. from the base of the tongue, but its anterior border was not so high. The tumor extended laterally as far as the alveolus, but not beyond the midline. It was slightly ulcerated on its surface, was almost of wooden consistency and was continuous with a mass in the floor of the mouth. The latter mass felt cystic. The submaxillary salivary glands and lymph nodes on both sides of the neck were visibly enlarged (Fig. 1) and were firmly attached to the deeper structures. The lower cervical lymph nodes were palpable, and they, as well as the epitrochlear, femoral, inguinal and iliac lymph nodes on both sides of the body, seemed larger than normal.

The tumor was evidently inoperable, with any hope of curing the patient, but his condition grew steadily worse while in the ward; on some days it was impossible for him to swallow. He had an evening rise of temperature to 100° F., and his white blood cells numbered from 15,000 to 19,000 on several occasions. It was finally determined to burn off the part of the tumor projecting above the tongue.

Operation was undertaken, December 12, with a tentative diagnosis of lymphoma of the lingual tonsil. Under intratracheal ether anesthesia, the lump was burned off the tongue by the electric cautery knife. Its base was almost of cartilaginous hardness. No bleeding occurred. Nothing was done to the mass in the floor of the mouth or to the submaxillary masses.

The patient improved rapidly after operation. His fever disappeared; he was free from pain and was able to swallow easily. The masses in his neck grew much smaller, and in less than two weeks they had contracted so much that wrinkles began to appear in the skin of the neck. He was kept under observation for several months and continued to do well. Eventually efforts to find him were ineffectual and, though no record of his death can be found in the vital statistics of the city of Philadelphia, it is not probable that he is still living.

The pathologic report showed that the sections of the tumor from the tongue contained lobulated areas of typical thyroid tissue (Fig. 2), which in places almost bordered the epithelial surface of the tumor. There was a very small amount of connective-tissue framework in the thyroid areas, yet more than in the normal thyroid. At one point in the section the epithelial covering was deficient, probably from ulceration.

In certain areas of the section, running from typical lobules of thyroid tissue, were dense accumulations of epithelial cells, placed

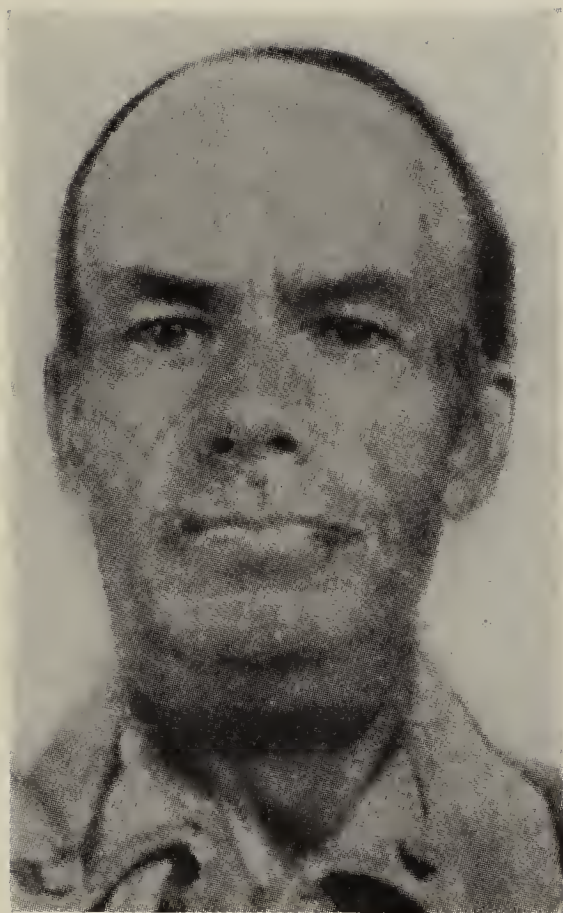


FIG. 1.—Nine days after excision (electric cautery) of tumor growing from right base of tongue (carcinoma of aberrant thyroid).

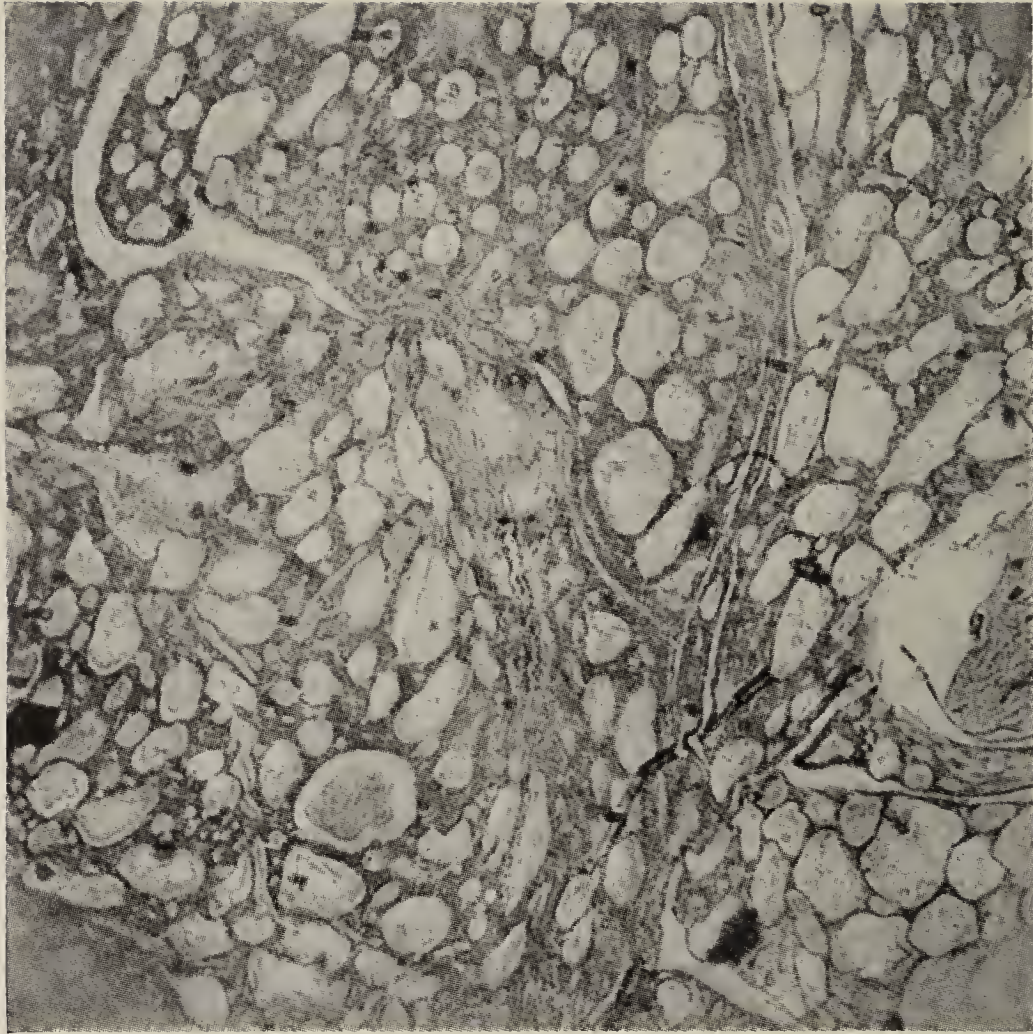


FIG. 2.—Lingual thyroid, showing carcinoma in other areas.

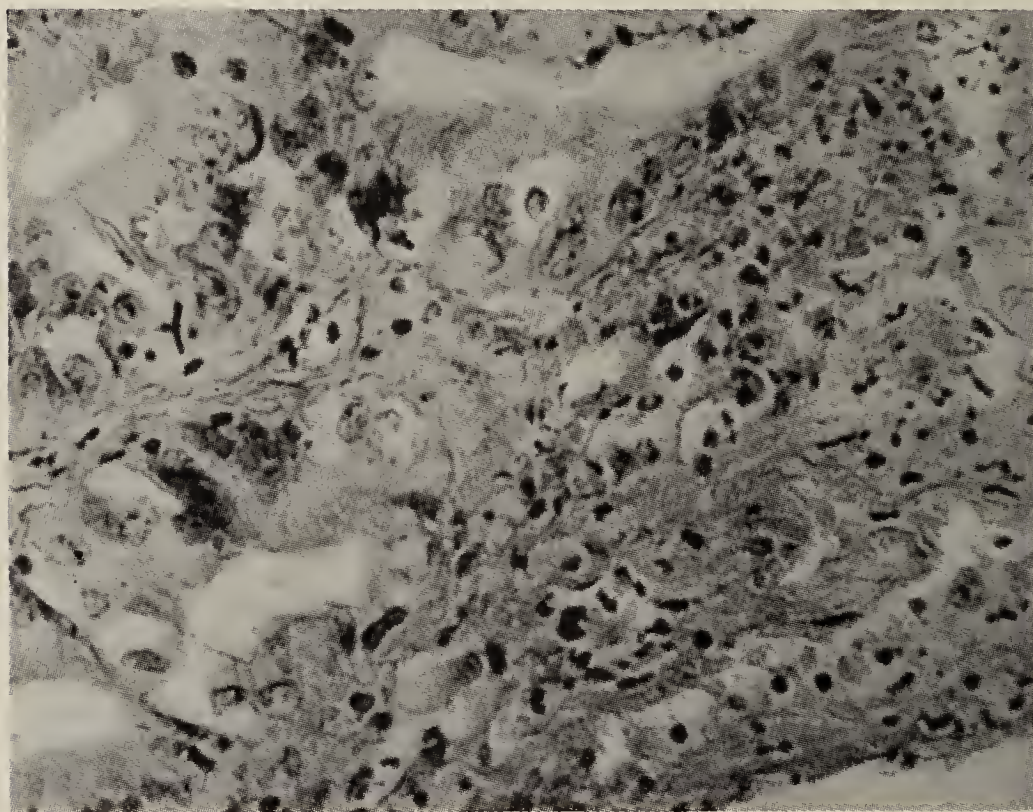


FIG. 3.—Carcinoma in lingual thyroid.

in bands or cords; and in still other areas there was a tendency to alveolar arrangement, that is, an arrangement typical of adenocarcinoma (Fig. 3). In other parts this carcinomatous arrangement was marked by increase of fibrous tissue, separating the epithelial cells into distinct cancer nests, with somewhat the appearance of scirrhous carcinoma. The carcinomatous areas bordered directly the epithelial covering of the tumor, causing thinning of the layers at certain points.

The diagnosis was adenocarcinoma of the lingual thyroid.

ACUTE PARENCHYMATOUS GLOSSITIS¹

WITH REPORT OF THREE CASES

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ACUTE parenchymatous glossitis is a rare disease. Many large hospitals have no cases upon records covering a long period of years. Only 3 cases have been observed in this hospital during the past twenty-two years. The literature upon the subject is fairly complete, however, probably because isolated cases have proven interesting. In December, 1921, 2 cases came under my observation within one week. These were essentially two different types in every respect, with 1 recovery and 1 death as the result.

The disease may be defined as an acute parenchymatous infiltration of the tongue. It is usually caused by some germ and is characterized by excessive swelling of the tongue or a portion of it, the process often progressing to suppuration, abscess or gangrene.

It occurs more frequently in males. The majority of cases fall within the age limits twenty to forty: but six months and eighty years are the extreme ages recorded. It is more prevalent during the winter months, when body resistance is lowered, and in the country rather than the cities.

ETIOLOGY. Exposure to cold and damp, especially when associated with a run-down physical condition and lowered resistance, is the most potent predisposing cause. There should be mentioned, also, as predisposing causes: injuries to the tongue, as by biting it; sticking it with a fork, fish or meat bone or toothpick; carious teeth; faulty dental work; foreign bodies held in the mouth; corrosives; hot liquids; insect bites; venoms; contact with cold metals; debauched states.

¹ Reprinted from Southern Med. and Surg. Jour., 1922, lxxxiv, 354.

Idiopathic cases are sometimes seen in scrofulous people, in convalescents from febrile diseases. Pepper mentioned that it was especially imminent after influenza, but this does not appear to have been borne out during the epidemic of 1918. Chewing certain acrid plants, eating certain foods, as celery and shellfish, are to be mentioned. Ambrose Paré reported two deaths from glossitis following the drinking of a vinous potion impregnated with saliva of a toad (Pepper).

Occasionally glossitis occurs during an attack of scarlatina, variola, scurvy, typhoid, glanders or septicemia. More frequently it is secondary to diabetes, rheumatism, mercurial poisoning, tonsillitis, pharyngitis and toxemic stomatitis. Occasionally it is associated with gastritis or epithelioma of the tongue.

Although the disease is usually an infectious one, the specific organism cannot always be determined. Staphylococci and streptococci have most frequently been recovered in pure culture from selected cases. Various other bacteria which normally inhabit the mouth have been blamed. Usually the mouth and tongue are naturally quite immune to germ infection. When body resistance is lowered, or when the organisms are injected into the tissues beneath the protecting epithelium, normal oral bacteria become pathogenic. Normally inert, they but await a suitable soil upon which to develop.

Development by extension has been pointed out by Prenn. Follicular abscess of the lingual tonsil may, by extension through the lingual follicles, transmit infection and thus be caused to form abscess within the tongue. Occasionally nasal operations, submucous resection, have been followed by acute glossitis.

PATHOLOGY. The process is essentially a violent inflammation with violent lymph and round-cell infiltration and swollen lymph vessels. In idiopathic cases it is believed to be merely an edematous phenomenon, not an inflammation. The connective tissue in the tongue is not very abundant; hence swelling may be almost unlimited. The infiltration is chiefly in the intermuscular substance. Damage to the parenchyma is, therefore, due principally to direct pressure upon muscular tissues. In cases advancing to suppuration, abscess or gan-

grene, muscular fibers are destroyed in proportion to the severity of the inflammatory process.

The inflammation may involve the entire tongue or merely a portion of it. The portion anterior to the lingual V is more commonly affected. There is a fairly definite connective-tissue septum running through the midline, which, however, becomes thin near the tip. In many cases this offers an effectual barrier to invasion of one-half the tongue. This explanation of why one-half the tongue may alone be involved is not universally accepted: Duckworth regards hemiglossitis as a catarrhal neurosis; Butlin believes it to be a parenchymatous inflammation of a superficial and benign type, differing from general glossitis only in the occurrence of a definite nodule, or lump, in the substance of the inflamed part. This lump, he explains, "may be present in many cases of general glossitis, but the excessive swelling prevents its being felt."

Cases have been reported where the attack was preceded by earache, applications to the tympanic membrane or disease of the middle ear. Such have been considered as nervous phenomena. Gueneau de Mussy produced violent swelling of the tongue by galvanizing the peripheral end of the lingual nerve, which ceased when the chorda tympani was cut. In such cases the swelling is not necessarily limited to one-half the tongue. Lewis reports a case where marked swelling of the tongue followed twelve hours after application of chromic acid to the tympanic membrane for chronic purulent otitis media. Another similar attack was noted within fourteen months. He attributed it to an angioneurotic edema from irritation of the chorda tympani nerve, which runs quite near the tympanum.

In infectious glossitis there is a difference in the staphylococcic and streptococcic types. When streptococci are causative there is characteristic local edema with danger of extension to the neck, glottis, lungs or pericardium. The infection may extend to the face and become typical erysipelas. In staphylococcic cases the swelling is localized, and there is little or no attempt to marked extension, unless there is a mixed infection with streptococci. Moreover, the formation of a well-localized abscess is usual.

When gangrene intervenes, it is usually due to pressure upon the tongue by the teeth. It is ordinarily limited to the portion of the tongue which is protruded beyond the teeth. Rarely gangrene affects the intraoral portion of the tongue or involves it in its entirety. The affected portion may slough away, leaving a clean base and subsequent cicatrices. Frequently violent hemorrhage follows sloughing.

SYMPTOMS AND DIAGNOSIS. The onset is usually so rapid that the clinician may not see the patient until the clinical picture is complete. There are, however, certain prodromal symptoms. Usually there is a mild sore throat, rather acute lingual pain, neuralgic pains in the back of the neck, ears and temporal regions, obviously due to the sensory distribution of the lingual and glossopharyngeal nerves. The lingual supplies sensation to the anterior two-thirds of the tongue and connects with the auriculotemporal branch of the trigeminal. The glossopharyngeal, which supplies sensation to the posterior one-third of the tongue, sends off a tympanic branch (Jacobson's nerve).

The swelling of the tongue, which is characteristic, may come on gradually or quite suddenly. Usually it reaches its maximum in twenty-four to forty-eight hours. Sometimes the prodrome is extended through ten days, without definite swelling of the tongue. The swelling may appear quite suddenly, as within two hours. In such cases the onset is usually at night. The patient may awake to find his tongue immensely swollen. Whether one or both halves be involved, the tongue will generally be so swollen that it almost completely fills the mouth. A part may be forced outside. The intraoral portion is dark red and moist, while the protruding portion is dry and livid, circulation being largely checked by pressure of the teeth. There may be teeth imprints and erosions upon the tongue.

Upon palpation, the tongue is found firm and uniformly as "hard as wood." It may pit a little on pressure. Sometimes there may be one or more points of resistance, and occasionally there is a soft point of fluctuation on the upper or under surface. This is especially so in hemiglossitis. However, this may not appear until the inflammation is well advanced and abscess formation has resulted.

Generally there is slight enlargement and tenderness of the submaxillary lymph nodes. One or both sides may be involved. Occasionally they are very much enlarged, but very rarely suppurate. When the posterior half of the tongue is involved the cervical nodes may be affected also.

The constitutional symptoms are quite characteristic. Pain is variable. It is spontaneous in some cases, absent in others. Where there is no spontaneous pain, marked pain may sometimes be provoked by slight manipulation of the tongue or even by touching it. Occasionally it is entirely absent. It is usually described as violent and shooting. It may be definitely localized or shifting from one side of the tongue to the other. There may be marked trismus, especially when the examiner pushed upon the mandible. This is due to contractions of the masseters, with rapid inhibition following encroachment upon the painful tongue. The tongue may be serrated against the teeth. The rugæ are usually effaced.

The swollen tongue makes speech unintelligible and pain serves as a check upon attempts. Early in the onset the speech becomes very nasal, as in marked angina or pharyngitis. Alongside this there is marked difficulty in mastication and deglutition. At the height of the attack even liquids may not be swallowed. There is usually interference with breathing, less marked where only half the tongue is involved than in total glossitis. The difficulty may be due to one or all of three things: (a) mechanical pressure of the swollen tongue upon the glottis and soft palate and pressing down of the epiglottis; (b) edema of the glottis; (c) a momentary nervous suspension of the act of respiration. Accordingly there are usually cyanosis of the lips, injected eyes, engorged face and a picture of terrible anguish. Suffocation rarely occurs, though sometimes tracheotomy is necessary. Sometimes the neck is so swollen as to injuriously press upon the jugular veins (Pepper).

The patient is usually quite restless. He dribbles saliva freely. Whether there is an actually increased flow of saliva has not been determined. Some consider the overflow as merely the normal amount secreted but which is unswallowed. The general symptoms are not particularly characteristic. Usually the fever ranges from 99.8° to 104° F., the pulse

being rapid and more readily compressible than normal. There is loss of appetite, insomnia and sense of suffocation. There may be constipation. Where erosion, suppuration or especially gangrene occurs, there may be diarrhea and toxemia, due to the passing of toxic products or decomposed tissue into the gastrointestinal tract. Occasionally there may be delirium, convulsions, prostration. Duperier explains that cerebral congestion is, however, not a symptom of glossitis, but rather a complication.

In favorable cases the symptoms begin to abate within three or four days. Rarely are they protracted longer than eight days. Bennett cites one case which lasted ninety days. Resolution is indicated by return of moisture to the tongue, progressive detumescence, subsidence of pain and dyspnea. The constitutional symptoms quickly disappear and phonation and deglutition are restored.

In suppurative cases local distress is usually protracted for about a week. Pain is more severe. Abscess may be suspected but remain undiscovered. In such cases spontaneous rupture and exit of pus quickly leads to recovery.

In cases progressing to gangrene there is a livid appearance of the part undergoing mortification. This may be localized or diffused. Gangrene is indicated also by "marked adynamic symptoms upon the part of the constitution," according to Bennett. A characteristic odor accompanies gangrene.

Certain conditions which cause a swelling of the floor of the mouth may closely simulate glossitis. Acute infection, salivary cysts, phlegmon and tumors may be mentioned. As a rule in these cases the pain and swelling of the tongue itself are not very marked. The chief point of differentiation is this: in glossitis the tongue is affected; in phlegmon of the floor of the mouth (Ludwig's angina, etc.) the tongue is not affected, but the floor of the mouth may rise above the alveolus. Moreover, in glossitis the tongue is pushed forward and is projected beyond the dental arcade, while in affections of the floor of the mouth the tongue is pushed upward and backward. Thebaud points out that in phlegmon of the buccal floor the swelling is chiefly "sushyoidienne," while in glossitis the swelling is chiefly within the buccal cavity.

TREATMENT. In all types the initial treatment is to give a saline purge. A certain number quickly recover without further treatment. This is especially true of the noninfectious types. It is, therefore, the part of wisdom to determine the causative factor if possible. Long, deep incisions are unnecessary in some of the noninfectious types of glossitis. Butlin states that in hemiglossitis the treatment consists of a purge, followed by a drink of chlorate of potash, liberal liquid diet, local applications of cold or, where warmth is more acceptable, warm gargles or bathings of the tongue with solutions of borax or potassium chlorate. It is doubtful if this idea is adhered to generally. Where pus is suspected, the treatment is surgical. The inquiring scalpel frequently finds pus where the diagnostic finger fails to appreciate localizing signs. An incision made over a fluctuating point is too frequently rewarded by evacuation of pus and rapid relief of symptoms to make medical treatment the treatment *par excellence*. Moreover, it would seem that, though the results may be ultimately as good in unoperated cases, yet the period of recovery is longer. Spontaneous rupture of abscess, which frequently occurs, is Nature's surgical treatment, but it is not devoid of danger, especially when it occurs while the patient is asleep. In some cases where a pus pocket is not incised the condition becomes chronic. Hahn reports a case where swelling of the right half of the tongue had recurred periodically for a period of two years. With each attack pus would be extruded from a sinus in mid-line in front of the lingual V. Recovery would follow in a few days. Finally, during an attack, a long, deep incision over the swelling and pack in the wound resulted in permanent cure. Demarquay cites 2 cases (of Blandin and Cousin) where diagnosis of cyst was held until incision proved the real condition.

Where both sides of the tongue are involved the treatment is essentially the same. Incisions along the dorsum of the tongue—one on each side 1.5 cm. from the median raphé about 2 cm. long and 1 cm. deep—should be employed. Unless the ranine vessels are injured there is little danger of hemorrhage. Free bleeding, which usually follows, is helpful. Relief is usually prompt, though the normal size of the tongue

may not be regained for some time. When there is localized hardness or abscess, puncture may be made, as in any abscess, over the localizing point.

The incisions mentioned may seem large when first made. It is surprising how small the incisions appear when the swelling has gone from the tongue. The wounds close quickly, heal rapidly, leaving merely a faint linear scar shortly, which in no way interferes with movements of the tongue or with speech.

When gangrene has set in, incise posterior to the gangrenous areas to allow the swelling to reduce. Relieve pressure upon the tongue by placing a wedge between the teeth or extracting the apposing teeth. Keep the tongue very clean by frequent washings. Butlin recommends for more vigorous treatment the application of 1 to 1000 perchloride of mercury, or pure carbolic rubbed in and afterward washed away. Iodoform powder may be dusted upon gangrenous sores. Care must be exercised that material and sloughs are not swallowed or allowed to run down the throat, as these are prone to cause autointoxication, intestinal upsets or septic pneumonia.

Occasionally involvement is so deep and so near the root of the tongue that the abscess is hardly accessible through the mouth. In such a case, as in one reported by Hahn, the incision and drainage must be made through the suprahyoid region. If suppuration occurs in the related lymph nodes they are also to be drained.

The constitutional symptoms may be improved by giving small doses of quinine and strychnine. Where pain is severe moderate doses of morphia may be used.

PROGNOSIS. While all cases are serious, the prognosis is as a rule favorable. In hemiglossitis, recovery is almost invariable. Where both sides are involved in a purely edematous or suppurative process, recovery is usual. In cases complicated by gangrene, prognosis is poor, recovery depending almost directly upon the resistance of the patient.

In a study of 145 cases, Bennett found record of 4 deaths (about 3 per cent). Of these 1 died during a debauched state; 1 of suffocation; 1 of paralysis of the palate, rupture of abscess, hemorrhage and collapse; 1 following tracheotomy. In reviewing a series of 32 uncollected cases appearing in avail-

able literature since 1910, there was only 1 death. This was in a case of gangrenous glossitis, complicated by aspiration, pneumonia and collapse, the report of which is appended (Case III).

In cases where there are frequently repeated attacks, prognosis is good, the ultimate cure depending upon the elimination of the causative factor. Bennett cites a case in which three attacks occurred in three years. Cure was finally effected by removal of amalgam fillings in some teeth. In another case there were six attacks. Removal of false teeth eliminated the disturbance.

In long-standing cases the danger lies in end-results. Bennett cites a case lasting ninety days in which marked adhesions formed on the tongue and cheek, which had to be severed and kept free. In only a very few cases, where very wide incisions must be made, or where gangrene occurs resulting in actual loss of tissue, is there the slightest interference with the use of the tongue or speech following convalescence.

CONCLUSIONS. 1. Acute parenchymatous glossitis is usually an infectious, inflammatory process, but may be a nervous phenomenon. It is frequently accompanied by suppuration, abscess formation and gangrene.

2. The entire tongue may be involved, but usually the swelling involves the portion anterior to the lingual V.

3. Where only one-half the tongue is involved, there is usually to be found a localized tumor tending to abscess formation.

4. While the symptoms are often alarming, and the prognosis to be guarded, the tendency is usually to recovery regardless of the treatment.

5. The best treatment consists of liberal incisions, oral hygiene, purgation and constitutional support.

CASE REPORTS. Observation I. Courtesy of Dr. T. R. Neilson. J. M., male, aged thirty-one years. Admitted to the Episcopal Hospital, March 26, 1920.

Present Medical History. Negative except several attacks of tonsillitis.

Chief Complaint. Sore tongue.

Began last Monday, March 22, 1920, with sore throat. Soreness cleared Tuesday. Returned two days later and tongue began to swell. Tongue very swollen, bright red and clean. There is no membrane. Tongue shows indentation of teeth. Throat negative. Speech very difficult. On admission the temperature was 104° F. within eight hours had risen to 105° F. Patient was given calomel; 2 grains, followed by magnesium sulphate. Potassium permanganate solution 1 to 4000 was used as a cleansing agent and potassium chlorate solution was used as an astringent mouth-wash. On the following day the swelling was greatly reduced. The temperature returned to 100.3° F. Pain was less marked upon swallowing. Patient continued to improve. The temperature dropped on the third day to normal. Patient was out of bed in five days. On the seventh day was discharged as cured.

Observation II (Service of Dr. A. P. C. Ashhurst). G. W., male, aged forty-six years. Admitted to hospital, December 1, 1921.

Past History. Erysipelas on nose and face one year ago, at which time was in bed for five weeks.

Present Illness. November 25, while eating fish at midday meal, he felt scratching on side of tongue. Thinks a bone stuck him. At 3.00 P.M. tongue began to get sore with feel as of something sticking in it. This continued until November 28, 1921 at which time a physician was called. A diagnosis of ulcer was made and it was "burned off with some liquid." Gargles were used frequently. The next day the same doctor advised that the man had an abscess of the tongue. November 29 the pain was so severe that he could not sleep. Speech became difficult. On account of the increasing size of the tongue he began gagging and choking. He could almost feel the tongue getting larger and larger. December 1, 1921, he was admitted to our service.

On admission the man was in great distress, but apparently not acutely ill. The mouth was partially open, the tongue protruding slightly. Upon examination it was found that the swelling, while marked, was limited to the right half of the tongue. At the junction of the middle and posterior thirds a mass 2 cm. in diameter, tense and yellowish, suggestive of pus, was seen. The floor of the mouth was not involved. The cervical glands were not palpably enlarged.

Treatment. Shortly after admission the patient was operated upon by Dr. Ashhurst. Under local cocaine anesthesia the tip of the tongue was caught with forceps. Incision was made along the right border of the tongue 4 cm. long, where the abscess was

pointing; 2 to 3 cc. creamy, greenish pus evacuated. Shortly calomel, 2 grains, followed by magnesium sulphate were given. During the night the patient was given morphine sulphate, $\frac{1}{6}$ grain. The following day improvement was marked. The swelling rapidly subsided. Five days after operation the tongue was very slightly swollen, and as the general condition of the patient was good, he was discharged as cured.

Observation III (Service of Dr. Ashhurst). W. C., female, aged sixty-seven years. Admitted December 8, 1921.

Family and past history negative.

Chief Complaint. Swelling of tongue.

Patient states that for some months she has been bothered with bad teeth; that at times those on the right, especially, have irritated her tongue. Five days ago she felt a little soreness in the left side of her neck. Later in the day the right side was painful and swollen. The following day the tongue began to swell rapidly. Yesterday it became so swollen that she could not talk. It was then that a physician was consulted. He advised her to come to the Hospital. She states that heretofore her general health has been very good.

Upon examination the patient was found to be a fairly well-preserved female, aged sixty-seven years. Complexion rather anemic, expression that of distinct distress. Temperature, 101° ; respiration, 28; pulse, 120. The mouth was open. The tongue was diffusely swollen and very red, covered with a greenish, mucoid and slimy substance, which had no particular odor. The tongue was so swollen that it protruded several centimeters beyond the teeth. The protruding portion was very dry and hard. Palpation was not painful, nor was there any area which was particularly soft or fluctuating. The tongue was scarcely movable. The speech was almost unintelligible. Swallowing was well nigh impossible. Blood-tinged saliva constantly dribbled from the mouth. The patient had to sit erect with head forward to breathe with any degree of comfort. The floor of the mouth was not involved. The teeth were in very poor condition. There were several sharp stumps, particularly in the lower alveolus. The glands in the submaxillary regions on both sides were somewhat swollen and hard. There was no cellulitis of the neck. Examination of the chest was unsatisfactory because of the dyspnea. No area of dullness was detected. Physical examination otherwise negative. The general condition appears to be poor, the patient apparently being almost exhausted.

Treatment. Operated on by Drs. Holloway and Crossan, one and a half hours after admission. One per cent novocain, applied locally. Incisions made on each side of median raphé 5 cm. long, 2 cm. deep. Incisions made on each lateral margin of the tongue about 1.5 cm. deep. A little pus was obtained from site of the left lateral incision, greenish and fetid. Rubber-tissue drains made to communicate between dorsal and lateral incisions. Silk suture passed through tip of tongue to hold tongue forward and to facilitate breathing. Several teeth were removed by Dr. J. R. Cameron, D.D.S., to relieve pressure on tongue. Bleeding was fairly free from incisions in tongue except on left lateral side where the cut area remained almost dry. As the patient's condition was very poor and verging upon collapse, it was thought unwise to purge her. Morphine and atrophine were given one half hour prior to operation. Following the operation continuous enteroclysis of glucose and soda was started. Strychnine, $\frac{1}{60}$ grain, hypodermatically every four hours and whiskey every four hours were ordered.

Results. On the following day the condition was very poor. The tongue was reduced somewhat, especially on the left half anteriorly. In this region the color had become darker, paler and yellowish. It looked as if it were undergoing mortification. No line of demarcation was detected. The respirations were less difficult and the patient was more comfortable. By 4 P.M. the temperature had dropped to 100° ; the respirations to 20, the pulse 90. However, the exhaustion was more marked. The patient was hardly able to endure lying flat in bed even on her side on account of dyspnea. She was too weak to sit erect or nearly so. Consequently some of the secretions and saliva must have passed down her throat. Swallowing was almost impossible. Dribbling from the mouth continued.

On the following day December 10, 1921, the tongue had subsided considerably, so much so that it barely protruded beyond the teeth. The left half anteriorly was involved in a slough of the entire thickness of the tongue. Over the neck anteriorly was noticed an area of redness, sharply defined with slightly raised margins, irregularly outlined, extending from the chin to the upper border of the sternum and from the trapezius on the left to the posterior border of the sternomastoid on the right. No bullæ or blisters were present. Erysipelas was diagnosed. The general condition was much worse. The temperature had risen to 102.4° . Stimulants were continued.

After midday collapse was imminent. Diffuse, bubbling râles were heard over the lungs. The temperature dropped to 100.2° F.

by rectum. The patient was conscious but paid little attention to surroundings. The temperature rose in the afternoon to 104° F. by axilla. Further treatment was without avail. The patient died shortly after midnight. General autopsy was not permitted. Portions of the tongue were removed after death. A specimen from the right side showed acute suppurative inflammation, that from the left, gangrene. Cultures were not made.

It may be admitted that this case was too far advanced before receiving surgical attention to hope for a successful issue.

TABULATIONS OF 32 CASES OF ACUTE PARENCHYMATOUS
GLOSSITIS REPORTED SINCE 1910

<i>Sex</i>	
Male	19
Female	8
Not stated	5
	<hr/>
	32
<i>Site</i>	
Both sides	21
Left half	8
Right half	3
	<hr/>
	32
Shortest time recovery noted	24 hours
Longest time recovery noted	26 days
Cases treated surgically	14
Cases not treat surgically	17
Cases not treated (refused)	1
Result—Known recovery	30
Death	1
Cases presumably neurotic	4

NOTE.—Since writing this paper von N. Mander, Copenhagen,¹ reports a case of edema of the tongue following the intravenous injection of mirion in a tabetic patient who had never had any syphilitic manifestations. Edema involved tongue and both submaxillary regions. Recovery followed within two days, without operation.

REFERENCES

- Baldwin, W. H.: Cincinnati Med. Jour., 1896, vol. ii, p. 143.
 Bennett: Wash. Med. Ann., 1906-1907 vol. v, p. 267.
 Butlin: Diseases of the Tongue, Cassell & Co., London, 1900, pp. 66-81.
 Cananaugh, John A.: Ann. Othology, Rhinology and Laryngol., St. Louis, 1918, xxvii, 206-212, Disc. 394-397.
 Dabney, Virginius: Jour. Am. Med. Assn., Chic., 1917, lxviii, 1476.
 Duckworth: Liverpool Med. Chi. Jour., 1883, iii, 195.
 Duperier: Thèse de Paris, 1906-1907.
 Fiorovanti: II Morgagni, 1914, lvi, 274.
 Gerhard, S. H.: Codex Medicus, Philadelphia, 1896-1897, iii, 421.

¹ Wiener Klinische Wochenschrift, March, 13 1922, p. 297.

- Greene: Med. and Surg. Reports, Philadelphia, 1891, lxxv, 333.
Hahn: Arch. Italiano d. Othologia, 1914, xxv, 57.
Hill, Berkley: Brit. Med. Jour., October 7, 1882, p. 683.
Lewis, Robert.: N. Y. Med. Jour., October 9, 1897, p. 494.
Loeb, Virgil: Dental Summary, Toledo, 1910, xxv, 668-673.
Lubman, Max: N. Y. Med. Jour., 1916, civ, 1146.
Pepper: System of Medicine, vol. ii, p. 359, Lea Bros., Philadelphia, 1885.
Prenn, Joseph: Boston Med. and Surg. Jour., 1916, clxxiv, 161-163.
Raynor, Frank C.: Laryngoscope, April, 1915, xxv, 227-228.
Ref. Handbk. Med. Sciences (Edited by T. L. Stedman), William Wood, & Co., N. Y., 1917, p. 209.
Stengel and Fox: Pathology, 6 ed., W. B. Saunders Company, Philadelphia, 1919, p. 598.
Summers, B. E.: Old Dominion Jour. Med. and Surg., Richmond, 1915-1916, xxi, 75.
Thibaud: Thèses de Paris, 1893-1894.
Thomas: Marseille méd., 1905, xlii, 512-516.
Wagner, Clinton: Med. Record, 1893, xliv, 523.
White, J. V.: Jour. Mich. State Med. Soc., Grand Rapids, Mich., 1916f, xv, 114-115.

SCIRRHUS CARCINOMA OF THE THROAT.¹

By WILLIAM R. WATSON, M.D.

OTO-LARYNGOLOGIST TO THE HOSPITAL

CANCER and sarcoma of the throat and tonsil are frequently encountered, the sarcoma being found, roughly speaking, about three times as often as carcinoma. When cancer is found it is usually of the epitheliomatous or squamous-cell type, with an occasional adenocarcinoma. Apparently, however, while comparatively common elsewhere in the body, notably in the female breast, scirrhous carcinoma is unique in that it is rarely found in the throat. I have covered the literature very thoroughly since 1893 until the present date without having found any report of a cancer in the throat of this type. Nor does Ewing, in his book on neoplasms, mention the existence of a tumor of this character either in the throat or tonsil.

The patient in this case is a young white woman, aged twenty-five years, whose family history is uninteresting. Her personal history, with the exception that she had had her tonsils removed when she was aged fifteen years, is also unimportant.

History of Present Illness. Her chief complaint was a swelling in the right tonsillar region which interferes with swallowing and phonation. Patient states that when her tonsils were removed in the Philadelphia General Hospital ten years ago she drained pus from the right tonsillar fossa for some time following operation. She also states that she could see a small lump in her throat from the time that her tonsils and adenoids were removed, but that it had given her no trouble until lately, when it began to grow large. It had been growing progressively larger for a year, until now it may be felt in the neck, and interferes with breathing, swallowing and speaking. Hearing in the right ear is also impaired.

This young woman was admitted to the wards of the Episcopal

¹ Read before the Otological Section of the College of Physicians, Philadelphia, April 21, 1926. Reprinted from *The Laryngoscope*, 1926, xxxvi 753.

Hospital on September 25, 1925, and on giving the above history she was referred to the laryngological service for opinion.

On examination, a hard nodular mass was seen externally, palpable below the angle of the jaw and inward almost to the mid-line. Intraorally a tumor was disclosed whose mass crowded the soft palate down to the left and so large as to almost fill the pharynx. The upper pole of the tumor reminded one of a very largely developed peritonsillar abscess, without the usual redness and edema. The surface of the tumor was smooth, covered by the anterior pillar and soft palate, and on palpation felt like hard rubber. By bimanual manipulation, with one hand externally and the fingers of the other pressing on the tumor within, the mass could be moved slightly from side to side, as if it were contained in a capsule. In other words, the opinion was formed—and correctly so, as it subsequently proved—that the tumor mass had not invaded the tissues outside of its own walls. The first natural thought was that it must necessarily be of a sarcomatous nature, although, personally, I could not accommodate my views entirely to this diagnosis, principally on account of the fact that it seemed to be encapsulated. All of the sarcomas of the throat with which I had come in contact had been fixed to the surrounding tissues. So, therefore, notwithstanding a history of lues was altogether lacking, this diagnosis was tentatively abandoned in favor of a gumma. Her Wassermann, however (three times repeated) was negative, and mixed treatment had no effect whatever in reducing the size of the tumor.

A biopsy was finally made, and on cutting into the tissue the knife slipped into a cavity, from which a bloody fluid escaped. This cavity was not large, perhaps about 1 cm. in width and depth, and the tissue which was sent to the laboratory was gathered from within the latter by means of a biting forceps.

The report from the laboratory was as follows: "The tissue shows an inflammatory exudate of a polymorphonuclear type, with an increase of connective tissue. Beneath this there is some suspicion of a tumor of undetermined nature."

This report being so unsatisfactory, a second biopsy was determined upon, and a specimen was cut this time from the body of the tumor away from the cavity and in tissue that was very hard and resistant to a very sharp knife. But the report in this case was also disappointing. All that the laboratory was able to find was a mass of fibrous tissue, with some evidence of myxomatous degeneration.

An enucleation of the tumor was finally decided upon, and in contemplation of this an *x*-ray of the chest of the patient was taken for the purpose of determining whether any metastasis had occurred.

The resulting opinion by Dr. Ralph Bromer is as follows: "Examination of the chest shows some slight peribronchial and hilum thickening, but otherwise no change. I do not think it shows any evidence of metastasis."

The tumor was removed on February 3, 1926, and the operating notes on her chart read as follows:

"An Eves knife was introduced under the anterior pillar at the junction of the latter and the base of the tongue and an incision made along the edge of the pillar until the base of the uvula was reached. This cleared the anterior pillar from the tumor. The tumor was then grasped with a tenaculum and pulled inward and downward, while, with a blunt dissector, the upper pole was delivered. The rest of the operation was done with the finger while making traction on the tumor with the tenaculum. There was a well-defined line of cleavage, or a capsule (call it what you will) for the tumor was stripped down and out of its fossa with comparative ease, leaving the fossa clean of any remaining tumor."

The tumor as it was removed from its bed had the exact appearance of a fibroma so often seen shelled out of the body of the uterus, and measured approximately 5 by 5 by 3 cm. There was one protuberance, almost pedunculated, which corresponded to the part of the tumor felt externally at the angle of the jaw and described as a "hard and nodular mass."

Report of the histology of sections taken from the tumor by Dr. C. Y. White, pathologist of the Episcopal Hospital, read as follows:

"Tissue taken from the tumor shows scirrhus carcinoma with the cells running in indistinct cancer columns. In some part of the tumor there are greater masses of cells in pockets, resembling a medullary type of tumor.

"The question arises as to whether this is cancer or sarcoma. From the description given above, I believe that the type of cell is one of epithelium rather than of connective-tissue type. The origin of such a tumor in the tonsillar tissue is, of course, difficult to state. The usual tumor of an epithelial character in this region is an epithelioma arising from the surface epithelium, or that which dips down in the crypts of the tonsil.

"The question of an alveolar sarcoma has been considered, with both hyaline and myxomatous degeneration, but the cell here found does not seem to correspond with the usual cells generally found in such tumors. In one section there is a tendency to glandular formation, so that such a tumor may have its origin in some of the epithelial structures in the vicinity of the tonsil."

With the idea of obtaining two opinions in the diagnosis of this case, sections of the tumor were subsequently shown to Dr. Baldwin H. Lucké, pathologist at the University of Pennsylvania, and he was inclined to concur in the diagnosis made by Dr. White.

After the operation and the diagnosis was determined upon, the question arose as to the prognosis in this case. Dr. Astley P. C. Ashhurst examined the patient some time after she had recovered from the operation and declared that he could feel her submaxillary and subparotid glands on the right and not on the left. On account of this he was rather pessimistic over her future: but when I reported the case at the staff meeting of the hospital last month, with the patient present, he failed to find the glands.

Since writing this article, February 7, 1930, I again saw this patient and found a growth in the same location as before, having the same general appearance and about one-quarter the size of the original growth. I fear that four years after the operation we now have a recurrence of the growth.

THE EPISCOPAL HOSPITAL AND THE GROWTH OF NEUROSURGERY

BY TEMPLE FAY, M.D.

NEUROSURGEON TO THE HOSPITAL

AND

NICHOLAS GOTTEN, M.D.

FORMERLY ASSISTANT NEUROSURGEON TO THE HOSPITAL

NEUROSURGERY as a special field is a comparatively recent addition to the family of surgical specialties which have evolved out of the practice of general surgery; so recent, in fact, that its recognition as a branch requiring special training has been accepted only since the World War, although its early inception numbered a few pioneers before 1914. The part which the Episcopal Hospital has played in the development of this new field is of particular interest.

It is rather striking that several of the outstanding figures who have made possible the present concepts and methods of neurosurgery, were closely associated with the Episcopal Hospital during some period of their activities, and hence the Episcopal Hospital has silently harbored within its walls the men and ideas which have contributed to the most remarkable advance of any branch of clinical science since the days of Hippocrates. In order to appreciate the influences which emanated from the Episcopal Hospital, it is necessary to trace the course of the development of neurosurgery through the period in which it had its beginning.

The records of the hospital show that as early as 1853 there appeared the diagnosis of "disease of the brain" in 2 patients, and "fracture of the spine" in 1 patient, who subsequently died. Unfortunately, neither histories nor hospital-treatment records of this period remain; we are left with only a record of the names, diagnoses and results of treatment. The list of surgeons on duty at that time included the names of

W. Byrd Page, H. H. Smith, Bernard Henry and Henry E. Drayton; Dr. R. A. F. Penrose was then an assistant surgeon and the first resident was Dr. Herman Robinett.

In 1857 the records show that 2 patients with concussion of the brain were treated; both recovered. There were 4 cases of paralysis and 2 of spinal irritation, with 3 cases of epilepsy under treatment. In 1858 the report showed 1 case of compressed fracture of the skull, along with other neurological cases which were variously diagnosed as "inflammation of the spinal marrow," "cephalalgia" and "spinal irritation." The methods and results of treatment obtained in the case of the compressed fracture of the skull are not known, but the surgeons of that period recognized the necessity of removing fragments impinging on the brain. The art of trepan was well established and skilfully used in cases of middle meningeal hemorrhage and brain abscess. In the time of Ambrose Paré, two hundred years before, trephining for abscess of the brain was attended with success when the cavity of the abscess could be opened. Having trephined over the abscess and opened the cavity, Ambrose Paré drained the cavity with a lead pipe made in the form of a blunt cannula, about the size of the index finger, with openings at the tip. This was tied in place and remained until healing occurred or the patient succumbed. Drainage of the cavity within the brain was further assisted by advising the patient to hold the nose, take deep breaths and strain forcefully, which produced an expulsion of the pus. Today we know that such a procedure produces a rapid rise of intracranial pressure, and this method of expulsion is probably not only unique but physiologically sound.

Dr. Samuel Ashhurst, who was surgeon to the Hospital from 1864 to 1881, trephined for freer access to the lateral sinus in a case of otitis media. This was "a decade before Zanzal, Horsley, Lane or Ballance had taken up the subject." Dr. B. Alexander Randall reported this early case at the meeting of the American Otological Society¹ in 1913, and is of the opinion that the operation must have been done before the year 1875, thus well antedating the earliest published work of

¹ Randall, B. A.: Trans. Am. Otol. Soc., 1913, xiii, 377.

other surgeons in this country or in Europe. The skull, showing the trephine opening, was deposited by Dr. Randall in the Mütter Museum of the College of Physicians of Philadelphia.

Dr. John Ashhurst, Jr., surgeon to the Hospital from 1863 to 1880, was the first to make clinically an accurate differentiation between the states of concussion of the brain, shock and the constitutional condition resulting from profuse hemorrhage. These states were confused, one with another, until Ashhurst's teaching appeared in the first edition (1871) of his *Text-book of Surgery*. Credit for this advance in surgical knowledge is given to Ashhurst by Prof. Edouard Quénu, in his monograph on *Toxémiê Traumatiquê* (Paris, 1919).

Dr. Charles B. Nancrede, surgeon to the Hospital from 1877 to 1889, was an outstanding authority on the surgery of the head during this period, and contributed an elaborate monograph on injuries of the head to the *International Encyclopedia of Surgery* (1881-1886), edited by Dr. John Ashhurst, Jr. Dr. W. Barton Hopkins, surgeon to the Hospital in 1884-1896, designed the cranial rongeur forceps which is still an approved instrument in neurosurgery.

In 1871 4 cases of fracture of the skull are recorded: 1 had a rupture of the middle meningeal artery; all of the patients died. In 1872 there were noted 6 cases of tic douloureux. In 1875 there were 8 fractures of the skull and 1 fracture of the vertebræ; all died. In 1876 2 cases were trephined for intracranial suppuration; both of these cases died, but the treatment was apparently similar to that which we utilize today, without, however, the knowledge of localizing factors which neurology has established for the neurosurgeon of today and which make an accurate localization of such an abscess possible. In 1884 we find records of 17 cases of fracture of the skull; 10 died; 1 patient was treated by trephining and recovered. In 1891 40 patients with fracture of the skull are recorded, 16 of whom died; among the 40 cases there were 13 trephines with 4 deaths. In this year are recorded 3 trephines for spinal injury, all of which recovered. In 1892 there were 8 cases of trephine with 1 death. At this time trephine was also done for Jacksonian epilepsy and 1 case of trephine for exploration of the brain with recovery was recorded.

In 1893 Dr. Charles H. Frazier became a resident physician and in 1904 was elected visiting surgeon. Since that time he has become one of the outstanding pioneers of neurosurgery throughout the world, and has done much by his care and technic in making possible the safe operative procedures of modern neurosurgery. He and Dr. William G. Spiller became engaged in the problem of section of the sensory root of the trigeminus for the relief of tic douloureux. The results of experiments on animals were the foundation for the operation which now bears the Spiller-Frazier name, and which is one of the brilliant achievements of neurosurgery. The development of a technic for surgery in cutting the sensory root of the trigeminal nerve was a contribution by Dr. Frazier which has opened up a field of possibilities hitherto unknown in the surgery of the brain.

To return to the records of 1895 we find 30 cases of concussion of the brain, with 8 deaths. Fracture of the skull is recorded in 50 cases with 15 deaths. There were 16 trephines for fracture, with 3 deaths. Here, then, is a great increase in the number of cases treated, with a striking decrease in the mortality.

In 1898 we find the record of a patient who was operated upon for brain tumor, but died. This year also marks our first case of neurectomy and neurotomy as well. One successful trephine of an epileptic, with recovery, is also noted. Trephine in 7 cases of fracture of the skull resulted in the recovery of the entire group. Four years later an excision of the Gasserian ganglion was performed, with recovery.

In 1905 there is a record of a case with an extradural hemorrhage of the cord, and one of hemorrhage within the cord. There were also 9 cases of trephine of the skull with recovery of all cases, and a case of brain tumor, which died. In 1906 a subdural hemorrhage is recorded with recovery; an extradural cord hemorrhage, neurorrhaphy and neurectomy for neurofibroma are also recorded. In 1906 the Spiller-Frazier operation of section of the sensory root of the trigeminus is reported in 1 case, with recovery.

It is well to pause here and survey the change which has come in the ten years from 1906 to 1916. During this time

more exact methods were applied to the subject of gross lesions of the brain and spinal cord. We find this reflected by an increasing number of cases relieved and a wider field of application of neurosurgery. During this period contributions on neurosurgical technic were being reported from various clinics in this country and throughout the world. The work of Ferrier, C. K. Mills, Sherrington, Kocher, Cushing, Sir Victor Horsley and a host of brilliant minds contributed actively to the knowledge of the central nervous system, and with this came the methods of operation which seemed possible after an understanding of the situation of the lesion.

Dr. William G. Spiller, who was associated with the Episcopal Hospital for a brief period of time, has probably made the greatest contribution toward the relief of suffering since the history of medicine began. It was Dr. Spiller's work which demonstrated the now-accepted method for the relief of trigeminal neuralgia (the most excruciating pain with which we have to deal). It was Dr. Spiller's discovery of the anterolateral columns in the spinal cord, and the function of these columns, which led him to suggest their section within the spinal cord for relief of intractable pain in the lower extremities. This operation, known as cordotomy, was first performed by Dr. Edward Martin in 1911, and this procedure has now become an accepted means for relief of pain. To attempt to enumerate the more than three hundred contributions to the medical literature by Dr. Spiller, the majority of them containing new and original observations which have had such a vast part to play in the developing of our present neurosurgical possibilities, would be impossible in a summary such as this.

At the present time our neurosurgical clinic is equipped to undertake all forms of neurosurgery, and the apparatus and newer instruments make its operating room one of the most complete in the country. The sympathetic coöperation of the superintendent and the nursing staff has made it possible to give to ward patients the special postoperative care so greatly needed in brain and nerve surgery, and the successful outcome of many of the cases operated upon has been directly due to this.

The traditions of the past, as well as the part which the Episcopal Hospital has played in developing this new branch of surgery, stands as an incentive to maintain the high standard of productive good which has arisen from the work of its former members, and to maintain a branch of surgery which the hospital helped to produce as a living tribute to those who were pioneers and responsible for its present possibilities.

TABULATION OF NEUROSURGICAL CASES OF EPISCOPAL HOSPITAL. (FROM YEAR 1852 TO 1928)

<i>Period Comprising 1852-1872</i>			
	Lived.	Died.	Total.
Brain abscess	11	1	12
Fractured spine	1	1
Spinal irritation	17	...	17
Fracture skull	11	9	20
Paralysis	2	...	2
Hemicrania	3	...	3
Neuralgia spine	3	...	3
Insanity	2	...	2
Hysteria	2	...	2
Inflammatory spinal marrow	1	...	1
Paraplegia	3	...	3
Paralysis sensory nerve	1	...	1
Convulsion	29	8	37
Local palsy	3	...	3
Cephalalgia	9	...	9
Meningitis	1	...	1
Spinal injury	1	1	2
Epilepsy	1	2	3
Concussion brain	6	...	6
Compound fracture skull	1	2	3
Concussion spinal marrow	1	...	1
Spina bifida	1	...	1
Myelitis	2	...	2
Fracture vertebra	1	1
Tic douloureux	6	...	6
<i>Period Comprising 1873-1893</i>			
Fracture skull	134	98	232
Fracture vertebra	1	1	2
Wound of scalp	7	...	7
Dislocation cervical vertebra	1	...	1
Concussion brain	81	19	100
Trephine abscess	30	15	45
Concussion spine	1	...	1
Luxation of servical vertebra	1	...	1
<i>Period Comprising 1894-1914</i>			
Cerebral concussion	38	2	40
Concussion brain	233	19	252
Fracture skull	294	155	449
Neurotomy	8	...	8

TABULATION OF NEUROSURGICAL CASES OF EPISCOPAL
HOSPITAL. (FROM YEAR 1852 TO 1928.)—(Continued)

Period Comprising 1894-1914

	Lived.	Died.	Total.
Fracture vertebra	6	6	12
Neurectomy	2	...	2
Concussion spine	2	...	2
Abscess brain	2	7	9
Excision of the fifth ganglion	2	...	2
Stretching sciatic nerve	1	...	1
Alcoholic injection	3	...	3
Alcoholic injury 5th	3	1	4
Neuritis	1	...	1
Thrombosis posterior sinus	1	1
Facial neuritis	1	...	1
Laminectomy—Gunshot wound	1	1	2
Laminectomy—Ant. poliomyelitis	1	...	1
Laminectomy	1	1	2
Trephine	116	35	151
Osteoplastic:			
Extra dural hemorrhage	1	...	1
Jacksonian epilepsy	1	...	1
Traumatic epilepsy	1	...	1
Cerebral hernia	1	...	1
Subpial hemorrhage	1	...	1
Gunshot wound	1	...	1
Trephine for fracture	34	2	36
Dislocated vertebra	1	2	3
Injection boiling water into cortex	2	...	2
Decompression—Brain tumor	1	1	2
Decompression—Fracture	6	...	6
Decompression and elevation of frag- ments	5	1	6
Craniectomy for fracture	9	2	11
Cerebral compression and contusion	93	4	97
Craniotomy abscess	3	3	6
Meningitis	1	1
Excision Gasserian ganglion	1	...	1
Extra dural hemorrhage, cord	3	...	3
Spinal cord hemorrhage	1	...	1
Subdural hemorrhage	1	...	1
Neurectasis	2	...	2
Excision neuroma	1	...	1
Neurorrhaphy	1	...	1
Neurorrhaphy laceration nerve	1	...	1
Brain tumor	1	...	1
Spina bifida	1	1

Period Comprising 1915-1928

Abscess brain	3	9	12
Concussion brain	125	4	129
Brain tumor	28	12	40
Extradural hemorrhage	1	1	2
Tic douloureux	3	...	3
Fracture skull	271	124	395
Fracture vertebra	19	11	30
Meningitis	1	1

TABULATION OF NEUROSURGICAL CASES OF EPISCOPAL
HOSPITAL. (FROM YEAR 1852 TO 1928.)—(Continued)

Period Comprising 1915-1928

	Lived.	Died.	Total.
Cord tumor	2	2	4
Trifacial neuralgia	7	...	7
Hydrocephalus	1	1
Laceration spinal cord	1	...	1
Torticollis	1	...	1
Intracranial hemorrhage	1	1
Concussion cord	1	...	1
Alcoholic injury (5th)	2	...	2
Compression neuritis	1	...	1
Raminectomy	11	...	11
Evulsion (subtotal)	18	...	18
Evulsion (total)	7	3	10
Gliomas	2	1	3
Endotheliomas	3	1	4
Angle tumor	11	...	11
Lipoma	11	...	11
Neurectomy	1	...	1
Cervical sympathectomies	3	1	4
Section of trigeminus	6	4	10
Decompression	2	...	2
Trephine	1	...	1
Craniotomy	58	12	20
Laminectomy	1	3	4
Chordotomy	3	...	3
Rhizotomy	4	4
Encephalograms	21	1	22
Supra-orbital neuralgia, avulsion	1	...	1
Traumatic myelitis	1	...	1
Brain atrophy	1	...	1
Craniectomy	1	4	5
Cervical rib	1	...	1
Ventriculo-encephalogram	1	...	1
Ventriculogram	1	5	6
Section glossoph. vagus and hypoglossal	2	1	3
Section ninth and sensory vagus	4	1	5
Ventriculoscopy	2	...	2
Intradural cord abscess	1	1
Callosal puncture	1	1
Trephine	2	2

OBSERVATIONS AND RESULTS FROM THE INTRACRANIAL SECTION OF GLOSSOPHARYNGEUS AND VAGUS NERVES IN MAN*

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NEUROSURGEON TO THE HOSPITAL

THE exact function of the glossopharyngeus and vagus nerves in man, especially as to their sensory values, has not as yet been definitely established. These two nerves are so intimately connected at their origin in the medulla oblongata, and after their exit from the jugular foramen, that experimental physiology has not as yet succeeded in isolating the functions of each. Pathological conditions frequently affect both of these structures simultaneously, not only in their central but also in their peripheral relations, giving rise to uncertain conclusions.

The observations noted in this paper have been derived from two cases: one in which the *intracranial root* of the ninth nerve was sectioned alone, at a point where its true components are assembled to compose the root, and are, therefore, the expression of the actual function of this nerve itself, before anastomotic fibers have been added from the neighboring vagus; in another case, the *intracranial root* of the vagus was sectioned alone and its manifestations studied.

This paper affords the opportunity of confirming in the human being the sensory determinations established by Sherrington¹ in the macaque. In fact, the means of study has been strikingly similar to his own by the use of residual areas of sensation after destruction of the adjoining sensory fields.

I have been unable to find any similar cases relating to man, where sensory studies have been made after careful section

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of the intracranial roots of the ninth and tenth nerves, with obliteration of the adjoining sensory fields also, by destruction of the posterior roots involved. I believe this to be the first presentation of sensory studies after isolated intracranial section of the glossopharyngeus and vagus roots in man, in this manner.*

Heretofore, information as to their function has been derived chiefly from pathological lesions situated near these two structures, but their separate functions have not been definitely established. Oppenheim² states regarding the glossopharyngeus: "Affections limited to this nerve have practically never been observed, and the vagueness which obtains as to its physiological position and its functions has not hitherto been elucidated by pathology. So far as I am aware, an injury limited to this nerve has not yet been observed." In speaking of the vagus he states: "It is usually affected in common with other cranial nerves, especially with the glossopharyngeal, accessory and hypoglossal."

Although an abundance of observations exists regarding extracranial involvement of these two structures, especially the vagus (Hirsch, Oppenheim, Kohnstamm, Lemon) by pathological processes and intracranial root involvement (Weisenburg³) as well as experimental evidence (Kriedl, Kohnstamm, Sherrington, Grabower), combined with surgical extracranial section (Sicard, Robineau, Adson), exact information regarding the part assumed by each of these nerves has not been definitely demonstrable. The reason for this uncertainty is evident from the intimacy of the structures as they enter the jugular foramen and pursue their early extracranial course. Individual involvement, except by careful surgical selection, is almost impossible.

Fig. 1 diagrammatically represents the relations and connections of these nerves as they leave the medulla and progress toward their peripheral destinations. Their communications and branches have been faithfully recorded from anatomical authorities such as Spalteholz,⁴ Piersol,⁵ Pitres and Testut,⁶ Zander⁷ and others. Anatomical relations in this diagram have not been maintained because of their complexity.

* See Addendum.

It is at once evident that section or destruction of either of these nerves, extracranially, produces an impure and misleading result because of a large branch given off from the vagus to the glossopharyngeus within the jugular foramen. The only certain means of determination lies in the careful selection of the roots as they emerge from the medulla to compose the intracranial portion of the root and thus represent the individual function of each. This has been possible because of the rapid advance of neurosurgery and the technic devised by Adson.⁸ It has been undertaken to relieve the extreme pain encountered in cases of metastatic carcinoma of the mouth and neck.

INTRACRANIAL SECTION OF THE GLOSSOPHARYNGEUS. The case (E. B.) is unique in the following respects: (1) isolation and section of the ninth nerve intracranially, without disturbing any fibers of the tenth or eleventh; (2) cervical rhizotomy of the second and third posterior cervical roots so as to produce an adjacent anesthesia of the head and neck; (3) section of the posterior root of the fifth, so as to produce an adjacent anesthesia of the face and forehead.

CASE I.—Mrs. E. B., aged forty-one years, was admitted to the neurosurgical service of the Episcopal Hospital, March 25, 1926. Referred by Dr. Eugene Pendergrass.

Diagnosis. Carcinoma at the base of the tongue on the left, with metastasis to the anterior cervical lymphatics.

Chief Complaint. Severe pain in the region of the left ear, behind the ear and extending to the vertex. Pain deep in the throat, greatly aggravated by eating and swallowing. Pain at times confined to the lower jaw and tongue. Only slight relief of pain even from large doses of morphine.

Operation. March 27, 1926. Cervical rhizotomy and intracranial section of the glossopharyngeus on the left (Dr. Fay). Recovery, with relief of all pain *excepting* that confined to the throat and ear.

Operation. June 26, 1926. Section of the posterior root of the trigeminus, on the left (Dr. Fay). Recovery. Pain in the throat and ear not relieved.

The result of the first operation (Fig. 2) was to produce only the same anesthesia which I⁹ have observed in other cases of cervical rhizotomy.

There was no demonstrable change in the area of anesthesia as a result of section of the ninth nerve, as well as the upper two cervical roots (C2 and C3). The zone of sensory supply attributable to the upper cervical posterior roots, as determined in a series of five cases by Spiller and myself,¹⁰ showed slight disturbance of pain perception as far forward as the hairline of the forehead, an overlap of one-half the area of the first division of the trigeminus. The extension from below over the angle of the jaw invaded the field of the third division of the fifth, and showed some slight disturbance of pain sense to the border of the second division. The distribution of pain sense was found to correspond more closely to the segmental pattern. The second cervical posterior root (the first is rarely present in man and when found is quite small) was found to overlap the trigeminal field and closely followed in its anterior limits the hair-bearing portion of the scalp and bearded area of the face, the posterior border overlapping into the field of the third cervical root.

Our findings are in close accord with Sherrington's¹ on the ape, and the anatomical dissections of Zander.⁷

The third cervical root zone shows a generous overlap from the fourth below extending for tactile sense as high as the angle of the jaw. The overlap for temperature is not as great as for light touch, and the overlap for pain is quite limited.

The region of the ear is of extreme interest. The auricle receives a quadruple sensory supply, and if the claims of some observers for the seventh and the ninth be accepted, there would be no less than six separate supplies for this structure. The *trigeminus* supplies the tragus, crus helicis, the anterior border of the helix and the anterior wall of the external auditory canal. This relationship has been carefully determined by Cushing,¹¹ Pegler¹² and many others, and corresponds to the findings in the above case. The *ramus auricularis nervi vagi*, demonstrated by Arnold, whose confines were established by Sherrington (Fig. 6) for the macaque, supplies chiefly the cavum conchæ and the cymba, extending its zone to include the anthelix, the antitragus and a portion of the lobus. The supply also includes a small triangle below and behind the lobe in the fossa anterior to the mastoid, and

extending on to the eminentia conchæ posteriorly, a finding which has been constantly present in our¹⁰ series.

The Second Cervical Posterior Root. This supply has not been definitely demonstrated in the human being, as no case has appeared where section of the fifth and tenth, and third and fourth, cervical roots has permitted the study of the residual areas of anesthesia. However, the work of Sherrington includes this zone of the external ear in the distribution of the second cervical root, and the dissections of Zander⁷ and Frohse¹³ indicate the presence of these fibers. We¹⁰ have been able to show slight hypalgesia over the posterior aspect of the eminentia scaphæ in the region of the fossa triangularis, as well as along the upper posterior border of the helix, but owing to the extensive overlap no anesthesia could be demonstrated.

The Third Cervical Posterior Root. When sectioned in conjunction with the second, there appears a definite area of anesthesia over the helix and the upper posterior aspect of the scapha, and extending into the posterior border of the anthelix (Fig. 3). Our¹⁰ observations have shown that the fourth cervical posterior root sends some fibers to the region of the helix, since after inclusion of this root the area became completely anesthetic for touch.

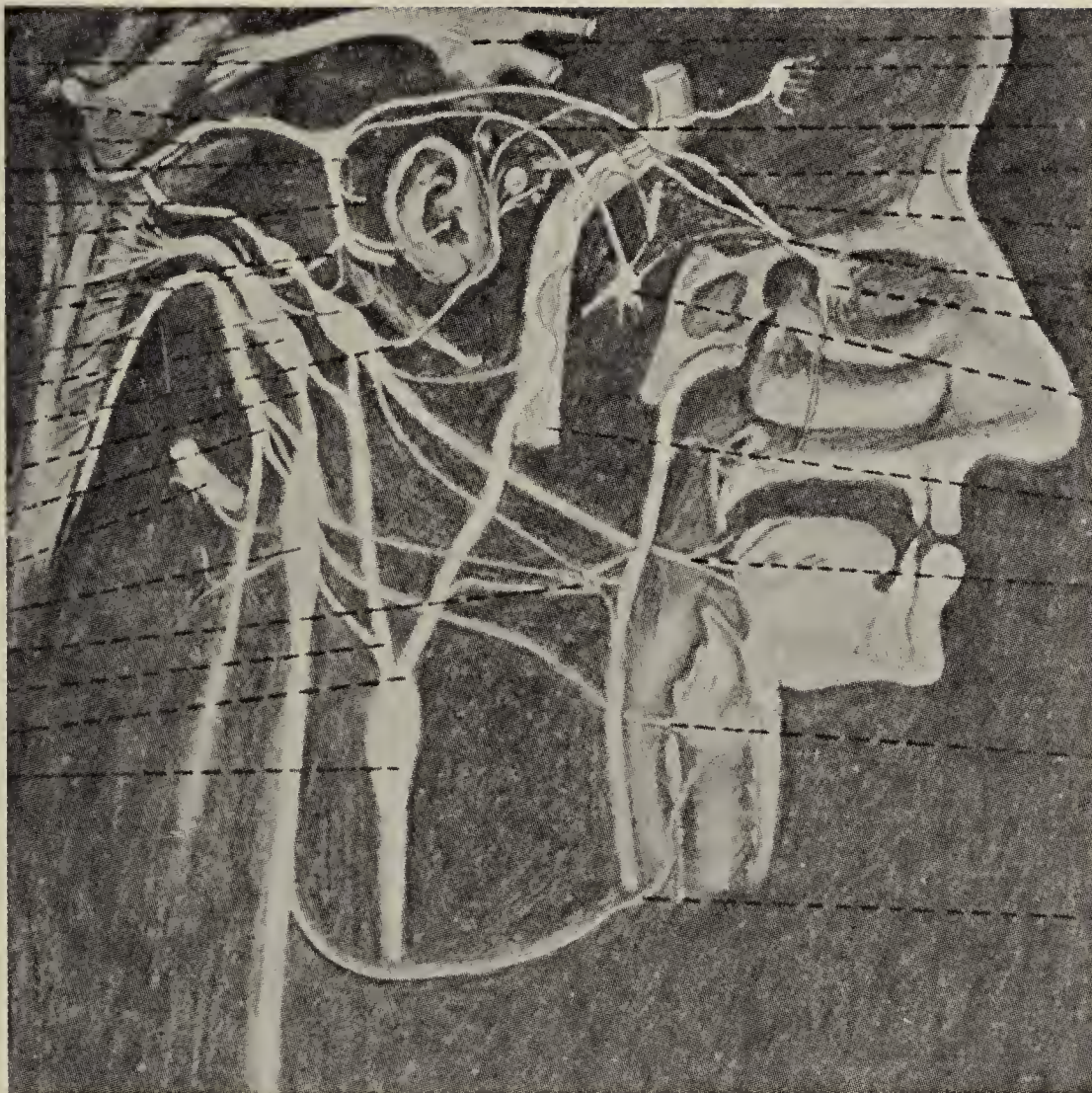
It is, therefore, evident in Case I that three of the contributing nerve supplies were destroyed, leaving the field of the vagus alone, which retained acutely all forms of sensation in the concha.

Section of the ninth nerve produced no demonstrable sensory change in the remaining field, and, as will be seen in Case II, ablation of the vagus showed no demonstrable sensory function attributable to the seventh or ninth, which remained undisturbed (Fig. 5) in this area.

These findings would seem to justify the conclusion in this case that the glossopharyngeal (intracranial root) supplies no cutaneous sensory elements to the region of the ear, face or neck.

I feel, after repeated careful studies, that these observations are convincing as regards the nonparticipation of the glossopharyngeus in a cutaneous sensory supply.

Portio major N.
Trigemini
N. Facialis
N. Intermedius . . .
Gang. geniculi . . .
N. Glossopharyn-
geus
Chorda tympani . .
Ramus auricularis
N. Vagi
Gang. superius . . .
Gang. petrosum . . .
Gang. jugulare . . .
N. Tympanicus . . .
N. Vagus
N. Accessorius . . .
N. Hypoglossus . . .
Gang. nodosum . . .
Plex. pharyngeus . .
N. Jugularis
N. Caroticus inter-
nus
Gang. cervicale
superius



Gang. semilunare
Gang. ciliare
N. Petrosus super-
ficialis minor
N. Petrosus super-
ficialis major
N. Petrosus pro-
fundus
N. Vidiani
Gang. sphenopala-
tinum
Gang. oticum
A. Carotis interna
N. Glossopharyn-
geus
N. Laryngeus
superior
N. Recurrens

FIG. 1.—Diagrammatic reconstruction of the seventh, ninth, tenth and eleventh cranial nerves, showing their intercommunications and the general distribution of their many peripheral branches. Accurate determination of the functions of each is only possible by section of the intracranial root, because of the addition of new fibers from one to another in their extracranial relations.

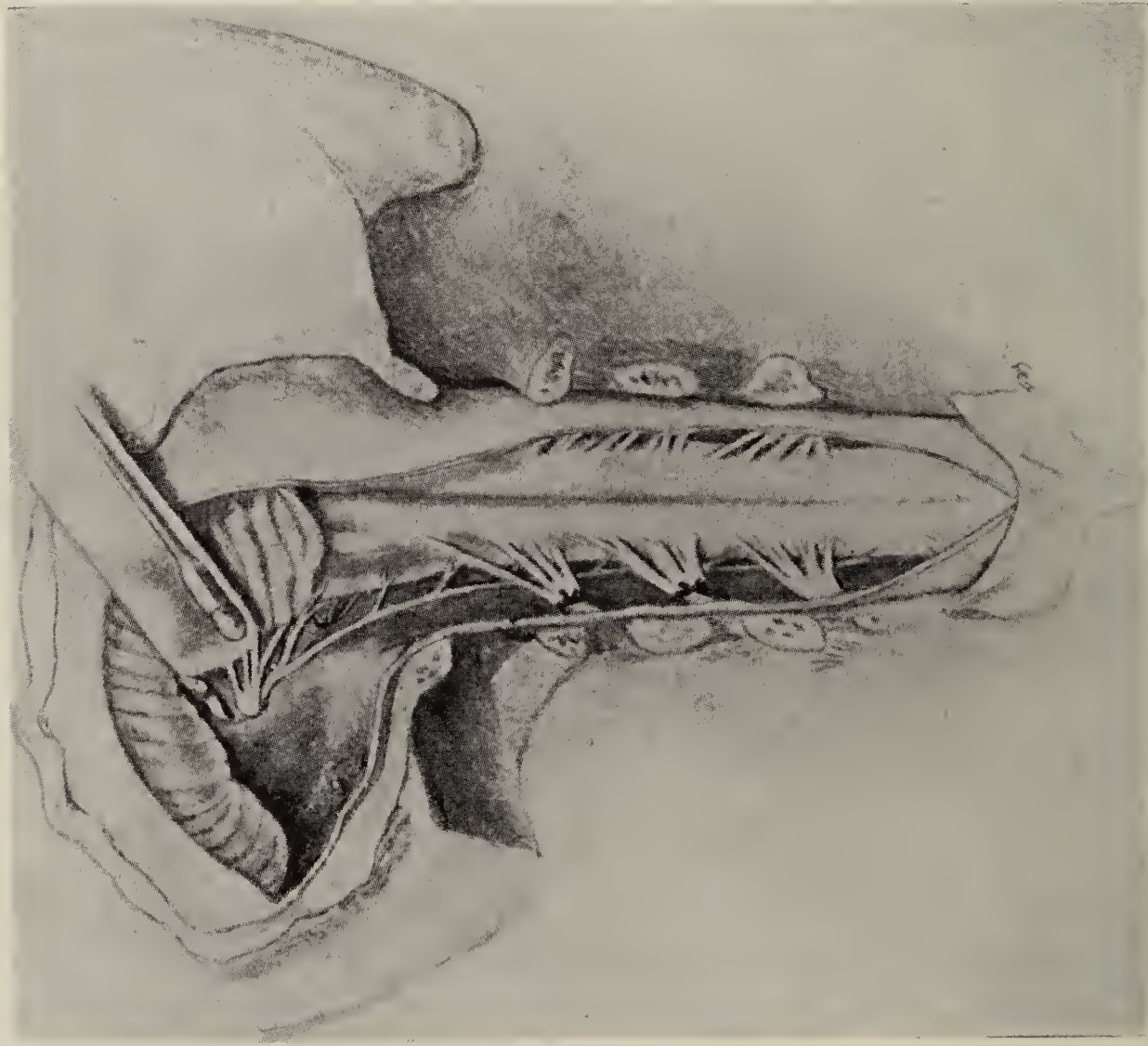


FIG. 2.—Operative approach for intracranial section of the ninth nerve, as well as for cervical rhizotomy (undertaken in Case I). Ninth nerve sectioned at its point of exit and the upper two cervical posterior roots crushed and ligated.

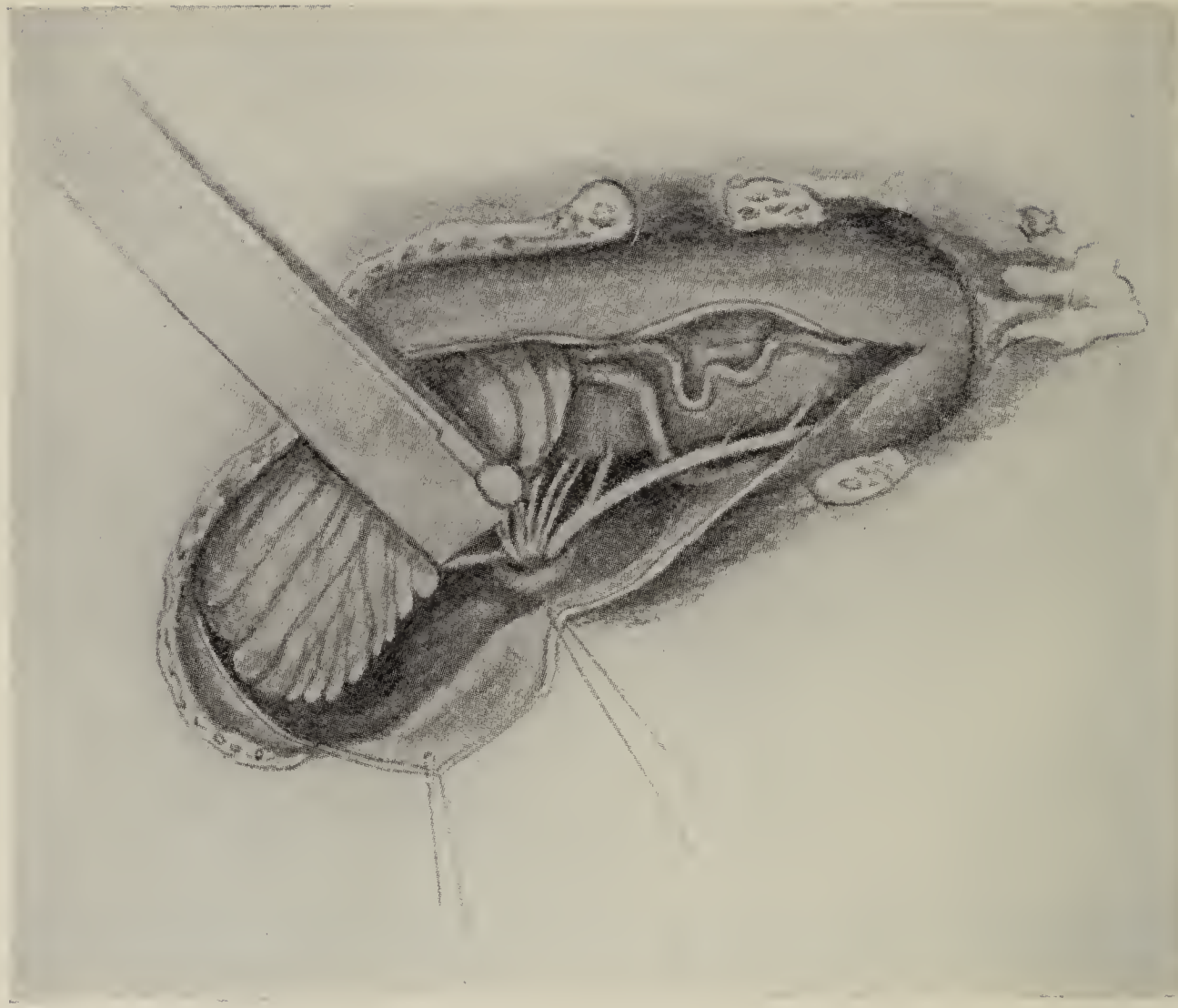


FIG. 4.—Intracranial approach to the vagus through a hemisuboccipital craniectomy and partial laminectomy. The fan-shaped fibers of the vagus can here be easily distinguished and sectioned at their point of exit without disturbing the ninth or eleventh (Case II).

Determination of the sensibility of the pharynx after section of the ninth was striking. No demonstrable change in touch, pain or temperature could be established. Unfortunately, this case was complicated by the primary carcinoma of the base of the tongue, involving the region at the base of the tonsil. Observations were difficult (pain in the ear and throat persisting), and definite conclusions must be avoided. The following observations seem justified:

1. Pressure against the pharyngeal wall and the introduction of a sharp object produced pain. This same reaction was obtained persistently after section of the trigeminal posterior root, the anesthesia from this operation including half of the tongue, anterior pillar of the fauces and roof of the mouth, but not the posterior margin of the soft palate.

2. The "gag" reflex remained intact.

3. The motility of the soft palate was impaired by the growth on the left, but there was no added relaxation or loss of alignment.

4. There was no added difficulty in swallowing or involvement of the swallowing reflex.

5. The sense of taste on the left, which had been present prior to operation, was lost over the posterior aspect of the tongue, and greatly impaired on the anterior lateral margin. Salt was recognized, but other flavors were not correctly appreciated and were frequently unobserved.

6. Salivation appeared on the left in increased quantity for several weeks.

7. The pain complained of in the throat and ear persisted, although all pain referred to the tongue, lower jaw, mastoid region and vertex was relieved, and it was possible to maintain the patient comfortably without morphine, by the use of allonal instead.

It would seem justifiable, perhaps, to assume from these findings that the glossopharyngeus in its intracranial root does not carry sensory fibers to the external ear; nor is there evidence that sensation of the common type is disturbed within the pharynx after its ablation. Its only demonstrable function has to do with the reception of taste impulses and, perhaps, the regulation of salivary and secretory responses.

Whatever sensory fibers appear in the extracranial or peripheral course of the nerve must be added to it from some neighboring source, undoubtedly chiefly from the vagus.

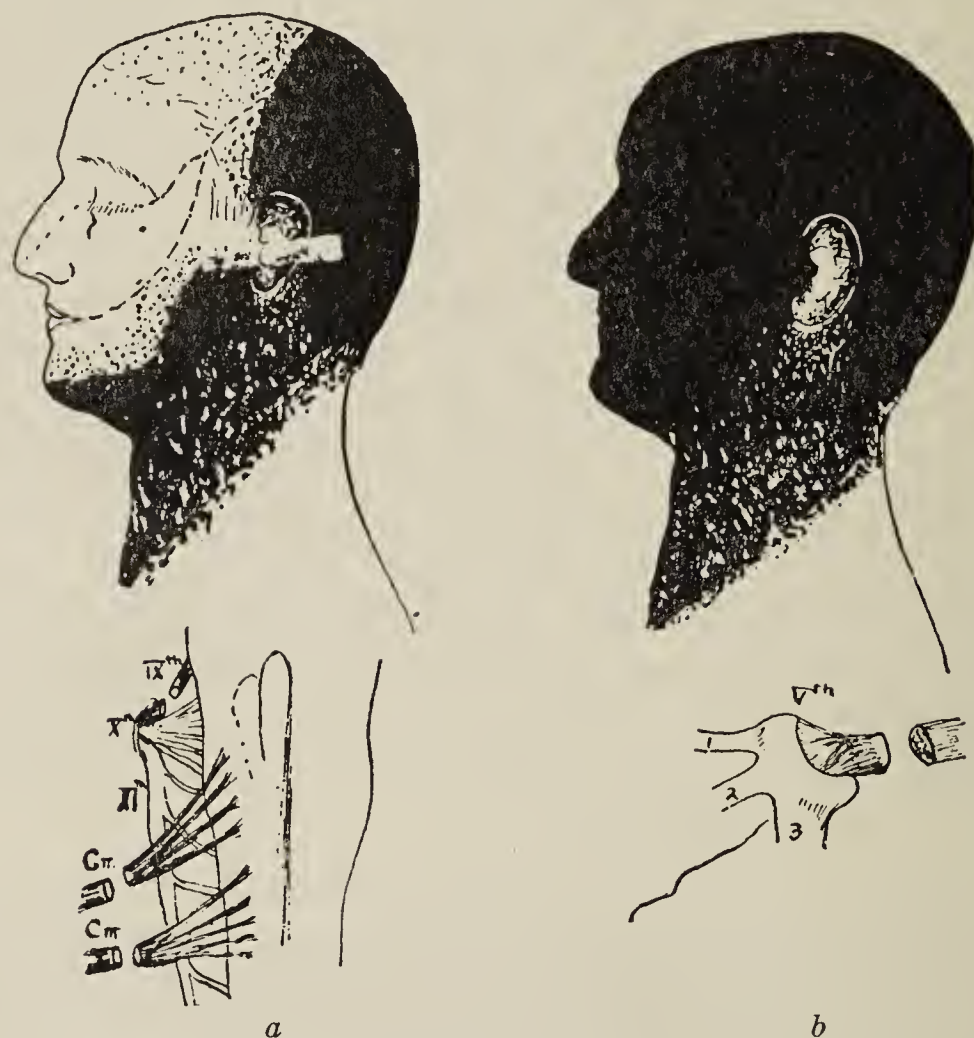


FIG. 3.—(a) Disturbance of sensation produced by the first operation in Case I. The area includes the dermatomes of the second and third cervical posterior roots. Note overlap of the fourth cervical root into the area of the third for touch sensation. The field of the second cervical root overlaps the trigeminal area to the hairline on the forehead and slightly on the cheek, as determined for pain sense by the comparative method. The findings are similar to those in a series of cervical rhizotomies, and there is no demonstrable zone attributable to the ninth. (b) Field of anesthesia after second operation, following complete destruction of the posterior root of the trigeminus. Note the residual area of sensation in the region of the ear.

INTRACRANIAL SECTION OF THE VAGUS NERVE. The next case presents even more interesting findings. I believe it to be unique from the standpoint of study in the following respects: (1) Subtotal avulsion of the posterior root of the trigeminus; (2) Intracranial section of the posterior roots of the vagus.

CASE II.—J. C., aged forty-eight years, was admitted to the neurosurgical service of the Episcopal Hospital, October 25, 1926. Referred by Dr. Eugene Pendergrass.

Diagnosis. Carcinoma of the tongue, with metastasis to the anterior cervical glands on the left.

Chief Complaint. Severe pain in the ear and throat. Pain in the lower jaw and tongue.

Operation. October 29, 1926. Subtotal avulsion of posterior root of the trigeminus, on the left (Dr. Fay). Recovery, with complete relief of pain in the tongue and lower jaw. No relief of pain in the ear and throat.

Operation. November 12, 1926. Suboccipital craniectomy; section of vagus roots on the left. Complete relief of all pain in the ear and throat. Paralysis and anesthesia of the pharynx and vocal cord on the left. Patient, sitting up, died suddenly from inhalation of fluid on the fourth day.

This patient also suffered from carcinoma of the tongue, with invasion of the floor of the mouth, and metastasis to the anterior cervical glands. His pain was confined to the throat and ear, as in the former case. He also had pain in the third division of the fifth nerve. The first procedure was the subtotal avulsion of the posterior root of the fifth nerve, supplying the area of the tongue and jaw involved by pain. It was possible to destroy the root of the third, as well as a portion of the second division, leaving intact the fibers of the first, so as not to endanger the trophic supply to the eye. This method of subtotal resection is that of Frazier.¹⁴ The patient recovered from the operation readily. The pain complained of in his tongue and lower jaw disappeared following the operation, and anesthesia was demonstrated in the third and in the lower two-thirds of the second division (Fig. 5). He still complained of the pain in the throat and ear, as in the case reported previously.

Because of the failure in the former case to relieve this type of pain by section of the ninth nerve, it was thought that section of the tenth nerve would bring about relief, in view of the findings in the former case, where, after section of the fifth, the ninth and upper cervical roots, this type of pain still persisted. It seemed logical, therefore, to attempt intra-

cranial section of the tenth. Two weeks after the original operation on the posterior root of the fifth, an intracranial approach to the vagus was made through the posterior fossa. As in the former case, a suboccipital craniectomy was performed under local anesthesia, exposing the cerebellum and lifting the cerebellar hemisphere on the side of the pain, exposing the jugular foramen, with the ninth, tenth and eleventh nerves emerging through it (Fig. 4). Careful selection was made of the tenth fibers only, and these were sectioned. During the sectioning of the vagus on the left side the anesthetist noted that the pulse dropped to forty. Following section, there was no further evidence of vagus stimulation.

The patient was carefully observed and repeatedly studied immediately after the operation, which was made possible as the operation was performed under local anesthesia. He was coöperative and showed no evidence of shock. Examinations on the second and third days were most satisfactory.

After carefully determining the area of anesthesia produced by section of the outer and lower two-thirds of the posterior root of the fifth, it was found that analgesia was complete over the tragus, anterior wall of the internal auditory canal, over the cheek in the third divisional area, and including almost but not quite, all of the conventional area for the second division. The tactile overlap from the cervical field was slight. Temperature areas conformed closely to pain, though they were slightly less distinct near the periphery (Fig. 5a).

Following section of the vagus root, several observations of extreme importance were made. The cutaneous area of anesthesia for the ear had enlarged from the base of the tragus and anterior wall of the internal auditory canal to include the posterior wall, the concha and, to a slight degree for pain only, the anthelix and antitragus (Fig. 5b). There was also a small area behind the ear, over the base of the scaphoid and in the region of the auricular mastoid fossa, close to the point of attachment, where all forms of cutaneous sensation were lost and pain sense greatly impaired in the neighboring field (Fig. 5c). Comparing this area with the residual zone remaining in Case I (Fig. 3b), there appears to be no doubt that the major supply to this area is by means of the vagus. The

findings are extremely like those of Sherrington on the macaque (Fig. 6).

It was striking to note the relief obtained from pain by the patient. He was profuse in his gratitude and insisted on sitting up in bed to express himself a few minutes after the close of the operation, in spite of instructions for absolute quiet following a posterior fossa exploration.

My experience has been that there is little reaction following this type of operation, where pressure from a tumor is not a complicating factor, if the procedure is done under local anesthesia, and the approach to the undersurface of the cerebellum is made extraärachnoidally, until the root is reached. Careful freeing of the arachnoid from the dura is possible, and if this be done no spinal fluid escapes from the cisterna magna into the wound and no blood is allowed to enter the sub-arachnoid space; failure to prevent this is, in my opinion, one of the causes for the postoperative crises seen in some cases of posterior fossa exploration.

The patient was immediately examined for motility of the pharynx and muscles of swallowing. The soft palate was seen to be paralyzed. There was great difficulty in swallowing, and the voice was very husky, due to paralysis of the left vocal cord. The patient choked on a small draught of water; he was carefully instructed in swallowing during the act of expiration, and after several attempts was able to take fluids by mouth, slowly, without choking or coughing. The "gag" reflex was found to be absent on the left. There appeared to be no recognition of pain or touch on the left side of the mouth, tongue, cheek (trigeminus), pharynx or base of the tongue (vagus). The pain had completely disappeared from the ear and throat; deep pressure on the left side of the neck against the mass had always produced extreme pain; he was no longer conscious of this procedure and, except for the mechanical difficulty in swallowing, there was no pain attached to the act. There was no further pain referred to the ear. Morphine was discontinued and he accepted the difficulty of swallowing and phonation without complaint. Relieved of his pain, he fell asleep and slept soundly for fourteen hours.

The following day he was examined again and the findings

carefully checked. The sense of taste was present on the left; he recognized salt, sweet and bitter substances, though he had difficulty in determining the position of the stimulus. The tongue and inner surface of the mouth were intact in this case;

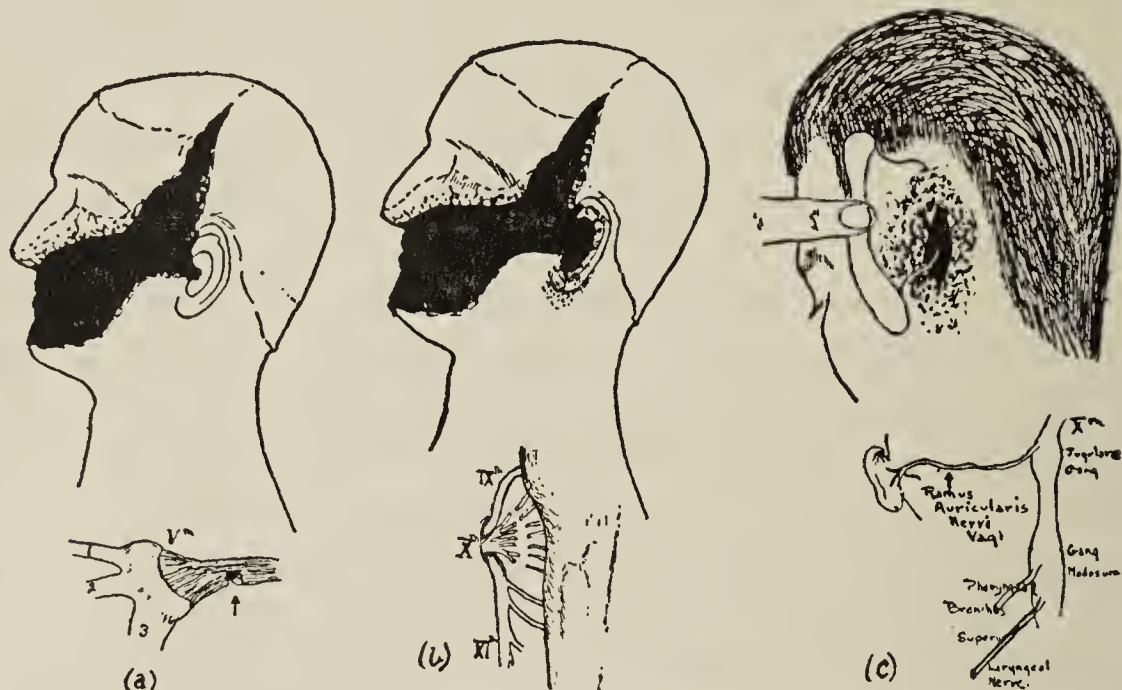


FIG. 5.—Case II. (a) Anesthesia produced after first operation, following subtotal avulsion of the posterior root of the fifth, but leaving intact the upper fibers, so as not to impair the trophic supply to the eye. (b) Resultant anesthesia after section of the intracranial root of the vagus. Note the extension of the zone of anesthesia into the concha and on to the anthelix. (c) Small triangle at back of ear, which became anesthetic following section of the vagus. This zone is supplied by the ramus auricularis nervi vagi. As all pain was relieved after section of the vagus, it is probable that the pain referred to the region of the ear arose from involvement of the pharyngeal and laryngeal branches of the vagus, with manifestations referred peripherally through the ramus auricularis nervi vagi.

the primary lesion, having been cauterized, had disappeared, leaving deep metastases in the floor of the mouth and throat through the lymphatics.

The nurse states that on the morning of the fourth day, as the patient sat up and taking a glass of water drank freely, he choked and died before she could summon aid. It is probable that he forgot momentarily the difficulty in swallowing and inhaled a large amount of liquid.

DISCUSSION. The two cases offer an extremely interesting physiological study, even in the presence of the pathological lesion. The infiltrating character of the lesion probably accounts for the lack of disturbance of the sensory function

prior to operation, and, aside from some hyperalgesia and the pain, there was no demonstrable neurological involvement of the areas under consideration.

Case I presented the possibility for isolated study of the tenth nerve in a surrounding field of anesthesia, as well as the loss of glossopharyngeal function.

Case II presented the possibility for isolated study of the ninth nerve in a surrounding field of anesthesia, as well as of the functions lost through destruction of the vagus.

These studies would suggest that the glossopharyngeal nerve, in its true root components, has to do primarily with the sense of taste, and in some way regulates secretory impulses arising from gustatory stimulation. It may well be considered as a nerve of special sense, similar to the optic and the auditory.

The vagus, in all probability, supplies common sensation to the pharynx and larynx, dispersing these fibers in their extracranial course directly by its own branches and indirectly by filaments carried by the extracranial trunk of the ninth. It also supplies common sensation to a portion of the external ear. It is undoubtedly the chief motor supply to the pharynx and larynx, and probably to the soft palate. It is responsible for the "gag" reflex and for reflex pain in the ear, due to involvement of its pharyngeal branches. The motor fibers which supply the vocal cord leave by the vagus root and not the vagus accessory, as has been claimed by some.

Section of the ninth nerve was undertaken in Case I because of pain in the throat and ear, a syndrome which has been described by Doyle,¹⁵ and given the name of glossopharyngeal neuralgia. Operative intervention for this type of pain has been undertaken successfully by Adson,⁸ their studies and work being based upon that of Sicard and Robineau.¹⁶ I owe to Adson the technic and inspiration for intracranial section of the ninth nerve. He first pointed out the possibility of intracranial section of this structure in 1923, and since that time has had three cases, the results of which will no doubt be published in the near future.

In his first case of intracranial section, he cut the ninth nerve along with a few fibers of the tenth. The sensory factors of this case were not published in the report of the

surgical procedure.⁷ In his second and third cases, the ninth nerve was sectioned; but, because of the condition of the patients, opportunity did not permit observations on the sensory results obtained.

Four cases of extracranial section of the ninth and tenth nerves for glossopharyngeal neuralgia were reported by Adson.⁸ One of this series, reported originally by Doyle,¹⁵ included section of the posterior root of the fifth, along with extracranial section of the ninth and the superior laryngeal branch of the tenth. The sensory findings were carefully charted. As is evident from the anatomical reconstruction shown in Fig. 1, the section of this nerve peripherally gives little information as to its individual root component, because of added fibers acquired upon leaving its cranial origin.

In the four cases of extracranial ablation of the ninth and tenth sensory factors, Adson states that in the first case the fifth posterior root was resected and the extracranial approach made to the region of the petrous ganglion of the ninth and the jugular ganglion of the tenth. The pharyngeal branches of the ninth and tenth were sectioned, with relief of pain in the throat. In the second case, the branches of the ninth were apparently not cut. The tenth was avulsed by mistake. In the third case, the branches of the ninth and the nerve itself were avulsed, but no final reports or sensory determinations were available, although the patient was free from pain. In the fourth case, both the ninth and tenth pharyngeal branches were avulsed, and pain developed in the eye with lacrimation and manifestations of rhinorrhea.

It is evident that Adson's work is the most extensive on this subject; but, as far as the determination of the actual sensory component of the ninth and tenth nerve roots is concerned, the four extracranial cases do not permit of accurate analysis, because of the possibility of fibers having been given off from the tenth to the ninth, as they leave the posterior fossa, and thus the combined supply destroyed.

The results of Adson's successful case of intracranial section of the ninth nerve have not as yet been published. I understood from a conversation with Doyle that the patient was relieved, but that section of the ninth had been attended by

some destruction of the fibers of the tenth, so that the relief of pain and the findings in this case are not clear-cut. Nevertheless, the relief of pain in this instance, as well as in the extracranial cases, points to the fact that the glossopharyngeus probably carries these sensory fibers in its extracranial course, or that the branches and roots of the vagus, which were sectioned, destroyed the pain arcs. Thus it becomes of great importance for future methods of relief to determine the exact functions of each of these structures, so that operative methods may be devised that will deal solely with the structures involved.

In my case, individual section of the ninth nerve intracranially failed to relieve the pain in the ear and throat, so that it was thought necessary to section the posterior root of the fifth nerve, believing that the auricular temporal branch of the fifth was responsible for the pain that occurred. Following section of the posterior root of the fifth, all pain was relieved *excepting* the pain in the ear and throat, which persisted and has persisted for the past thirteen months, during which time the patient has been under repeated study. Though the progress of her carcinomatous invasion has been greatly delayed by deep x-ray therapy, she is gradually becoming weaker and the growth is enlarging.

In both of these cases, pressure over the lateral aspect of the neck, in the submaxillary region and that of the superior laryngeal nerve, produced sharp pain in the throat and also pain referred to the ear. No sensory loss in the peripheral skin areas was present before operation. In both cases it was difficult to determine why the patient should have pain in the ear when the growth itself was situated in the floor of the mouth and in the upper portion of the neck. Unless we assume the extension of the carcinoma backward and upward, far beyond its confines as determined clinically, there appeared to be no explanation for the pain referred to the ear. However, after the destruction of the superior laryngeal portion of the tenth, as well as of the ramus auricularis nervi vagi, represented in the intracranial section of the tenth nerve, the pain disappeared.

It would, therefore, seem that carcinoma invading the floor

of the mouth is capable of referring pain to the ear through the distribution of the superior laryngeal nerve and the pharyngeal branches of the vagus. Irritation of these structures, both by pressure and the act of swallowing, produces a reflex pain through the same nerve, *i. e.*, the tenth, referring that pain cutaneously to the distribution of the ramus auricularis nervi vagi, within the ear.



FIG. 6.—The “completely delimited area of auricular of vagus,” determined experimentally for the macaque (Sherrington). Compare the areas obtained in man. (Figs. 3 and 5.)

From my observations, therefore, it would seem that the sensory fibers of pain, distributed to the pharynx and to the region of the ear, in the zone of so-called glossopharyngeal neuralgia, do not correspond to a function of the ninth nerve in its intracranial representation; this syndrome is in reality a manifestation of involvement of the tenth nerve in its sensory components. The term “glossopharyngeal neuralgia” does not designate either the exact location of the pain or the structures which transmit the painful impulses. A better term, inclusive of both factors, would in my opinion be “vagal auricular-pharyngeal neuralgia,” the manifestations of which are those of so-called glossopharyngeal neuralgia.

CONCLUSIONS. 1. The glossopharyngeus is probably a nerve of special sense, devoted to gustatory and secretory function.

2. The vagus nerve supplies a small cutaneous area in the region of the concha of the ear, as well as common sensation to the pharynx and larynx. It contains, in all probability, the major motor fibers which supply the soft palate and pharyngeal wall, and it is the motor nerve for the regulation and motility of the vocal cord. The “gag” reflex is a function of the vagus.

3. Intracranial section of the root should thus determine the nuclear functions of these nerves and, further, establish the exact physiology of each.

ADDENDUM.—Since the presentation of this paper and the submission of the manuscript for publication, Dandy (Arch. Surg., August, 1927) has published the results of two cases of intracranial section of the glossopharyngeus nerve in man, with a comprehensive review of the cases in the literature. The work was undertaken one year after the case reported herewith, and some months after the presentation of my findings before the American Neurological Association, so that his claim for priority must be denied. His article does not mention the details of his sensory studies, but simply ascribes to the glossopharyngeus an area for all forms of sensation, including taste, in the region of the posterior pharynx from the Eustachian tube to the upper border of the larynx, including the posterior third of the tongue and the epiglottis. These findings do not agree with those of Doyle's case or my own, and are not consistent with the neuroanatomy of the parts depicted. There is no mention of the zones of overlap or types of sensation involved. The "gag" reflex is not mentioned. In my experience it is almost impossible to chart the area of a single nerve supply to its correct limits in the presence of adjoining functioning fields that overlap, and the only true method would seem to be the one adopted by Sherrington, of blocking-out the neighboring supplies to determine the exact function of the desired structure.

BIBLIOGRAPHY

1. Sherrington, C. S.: Experiments in Examination of the Peripheral Distribution of the Fibers of the Posterior Roots of Some Spinal Nerves, Phil. Trans. Roy. Soc., London, Series B, 1898, cxc, 45.
2. Oppenheim, H.: Textbook of Nervous Diseases, Eng. ed., 1911, i, 495.
3. Weisenburg, T. H.: Jour. Am. Med. Assn., 1910, liv, 1600.
4. Spalteholz, W.: Hand Atlas of Human Anatomy, 4th Eng. ed., 1923, vol. iii.
5. Piersol, George A.: Human Anatomy, 1916, vol. ii.
6. Pitres, A., and Testut, L.: Les nerfs en schémas, Paris, 1925.
7. Zander, R.: Merkel's Festschrift, 1897, p. 1.
8. Adson, A. W.: Surgical Treatment of Glossopharyngeal Neuralgia, Arch. Neurol. and Psychiat., 1924, xii, 487.
9. Fay, Temple: The Surgical Relief of Pain in Deep Carcinoma of the Face and Neck, Am. Jour. Roent. Rad. Therap., vol. xiv, p. 1; Cervical Rhizotomy for Pain in Carcinoma of the Neck, Surg., Gynec. and Obst., vol. xliii, p. 366.
10. Spiller, W. G., and Fay, Temple: Observations on the Sensory Supply of the Head and Neck, Am. Neurol. Assn., June, 1926, unpublished.
11. Cushing, Harvey: The Sensory Field of the Cranial Fifth Nerve, Bull. Johns Hopkins Hosp., 1904, xv, 213.
12. Pegler, L. H.: Anatomical Tables of the Anatomy and Physiology of the Fifth Cranial or Trigeminal Nerve and of Its Ganglia and Connections, London, 1913.
13. Frohse, Fritz: Die oberflächlichen Nerven des Kopfes, Berlin, 1895.
14. Frazier, Charles H.: Subtotal Resection of the Sensory Root for Relief for Major Trigeminal Neuralgia, Arch. Neurol. and Psychiat., 1925, xiii, 378; Frazier, Charles H., and Whitehead, E.: The Morphology of the Gasserian Ganglion, Brain, 1926, xlviii, 458.
15. Doyle, J. B.: A Study of Four Cases of Glossopharyngeal Neuralgia, Arch. Neurol. and Psychiat., 1923, ix, 34.
16. Sicard, R., and Robineau: Algie vélo-pharyngée essentielle; traitement chirurgical, Rev. neurol., 1920, xxvi, 256.

FRACTURES: 1853 AND NOW

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THE surgeons of the three-quarters of a century preceding the establishment of the Episcopal Hospital left little or nothing for the surgeons of the next seventy-five years to discover in the art of diagnosis and prognosis of fractures. On the other hand, the year of birth of our hospital was the beginning of an era of development in the treatment of fractures, and the general surgeons of the past seventy-five years, with the aid of the radiographers, have almost converted the treatment from an art to a science.

ADVANCES IN THE ART OF DIAGNOSIS AND PROGNOSIS. The classification of fractures in use today is essentially the same as that which was in vogue in 1853. Acute clinical observation and repeated dissections had then established the existence of the comminuted, the oblique, the transverse, the longitudinal, the fissured, the serrated and the complicated fractures. The fractures occurring in the femoral necks of aged persons, as the result of trivial trauma were accurately described in Sir Astley Cooper's lectures, published in 1838. Colles' fracture and Pott's fracture were well known seventy-five years ago as typical varieties of fractures, and, also, it was common knowledge that fractures of the upper end of the radius were frequently complicated by dislocations of the ulna.

The one outstanding error in the fracture literature of that period is a too-great emphasis on the rôle of muscles in the production of deformity. Practically no attention was given to trauma as a factor in the displacement. For instance, the valgus deformity of ankle fractures was attributed to the action of the peronei. It is true, also, that fractures of the carpal scaphoid, or of the os calcis or of the spine of the tibia are not mentioned by any of the authorities in the middle of

the Nineteenth Century; but, on the whole, there was very little about the diagnosis of fractures which was not known by the surgeons of that period.

Then, too, the pioneer surgeons of the Episcopal Hospital could prognosticate, by virtue of the writings of Cooper, that the final results of a fracture of the neck of the femur would be nonunion. Cooper doubted that union ever did occur in these cases, and because of this he advocated the ambulatory treatment, advice which is again being propounded by some surgeons today. Also, one finds in the literature of that period frequent references to the union of fractures of the patella and of the olecranon "by ligament;" what we surgeons of today term fibrous union. The art of prognosis in fractures was a complete structure ere the foundation stone of the Protestant Episcopal Hospital was laid.

ADVANCES IN THE TREATMENT. Though it can be safely assumed that the treatment of fractures today is far superior to that of fifty or seventy-five years ago, one cannot compare the results in percentages of the good, fair and bad classes because end-result study is a comparatively recent development. However, an accurate index of comparison is afforded by a review of the literature of the earlier years and by consideration of the equipment of that period.

1853-1893: There are numerous references in textbooks and surgical lectures published during the early days of the Episcopal Hospital about bad results in fractures at the ankle; also, in compound fractures of the leg it was not then considered unusual, nor the result of poor care, to have the patients recover with $1\frac{1}{2}$ to 3 inches of shortening. Many patients with compound fractures lost their limbs from infection and quite a few lost their lives from infection or tetanus. In writing about fractures of the femur, Hamilton, in the year 1872, stated that it was common to obtain union with shortening, and, he added, "Those surgeons who claim otherwise through ignorance, inadvertence or intentionally . . . do not tell the truth." The bad results were not due to a lack of skill; the surgeons did not possess the necessary equipment.

Consider that the surgeons of 1853 had not become acquainted with the advantages of reduction under anesthesia;

anesthesia was not yet a decade old. Even for thirty or forty years after Bigelow's paper on anesthesia (1846), surgeons seldom used ether in reduction. Reduction in those days was a ceremony, a ceremony which required one assistant to make extension and a second assistant to counterextend, while the surgeons manipulated the bones in place. Unfortunately for the end-result, the ceremony was postponed until the inflammation subsided, and the "final adjustments" were made at a later period, or what was called the period of "nonreparation." We know today that immediate reduction is necessary because, as hours elapse, there is an accumulation of blood from the torn tissues which by its bulk, prevents extension of the muscles, and because, as days pass by and the clot becomes organized, the concomitant fibrous tissue will prevent extension of the muscles.

Reduction without efficient immobilization in most fractures is as bad as poor reduction. Even if the pioneer surgeons of the Episcopal Hospital and their confrères had secured good reduction, they had no efficient means of maintaining the fractured ends in position. For instance, in fractures of the femur immobilization was secured by a wooden splint extending from the axilla to below the foot, with indifferent extension from the ankle to the end of the splint (Liston's splint); with this apparatus it is not astounding that shortening was the usual result. Fractures of the leg and those of the ankle were treated by a wooden splint to the mesial aspect of the leg, with the foot bandaged in the varus position.

Though plaster-of-Paris bandages had been introduced into surgical practice in 1852, they were not widely used in fracture treatment until thirty or forty years later. It was contended that their only advantage was "rapid drying." What is so obvious today, that "rapid drying" is an aid in the prevention of displacement, escaped the men who were using bandages coated with shellac, or with gum arabic and whiting, or with white of eggs and flour. Nine years after the appearance of plaster, Buck published an account of his traction apparatus, to be followed in a few years by Hodgen's splint, which in turn was followed by the Hamilton splint in the late sixties and the Thomas splint in 1875. It is a safe bet that, with all four of

the latter methods, the disadvantages were condemned for many years before their virtues were extolled. All of the improvements between 1852 and 1875—the plaster bandages, Buck's extension, the Hodgen, Hamilton and Thomas splints—were the result of a development in the consciousness of surgeons that immobilization is as necessary as reduction.

The very bad results in compound fractures of that period were due solely to infection. These were the days of "laudable pus:" Lister's paper on the "Antiseptic Principle in the Practice of Surgery" appeared in 1867, but, like many new ideas in medicine, it had to make its way, and it was many years before Lister's ideas were adopted. Then, too, tetanus was thought to be a nervous affection until Nicolaier discovered the tetanus bacillus in 1884. Severe infections, setons, amputations and deaths were frequent experiences in compound fractures of that day.

The establishment of the Episcopal Hospital marks the dividing line between the new and old in the treatment of fractures. Within the first score of years of its existence there was developed efficient immobilization apparatus and a means of controlling infection. At the end of the second score of years (1893), a check-up on the new methods was afforded by Röntgen, when he discovered the *x*-rays. And so it can be said that the foundations for most of the methods of treatment we use today were laid between the years 1853 and 1893.

1893–today: Asepsis, tetanus antitoxin, specialization, propaganda and end-result studies are the contributions of the past twenty-five years to the treatment of fractures. Each item is a wedge by means of which fracture treatment has been driven from a lower to a higher plane.

Asepsis has prevented the horrible results in compound fractures. It has made operations on cases of nonunion or malunion, on compound fractures, on fracture of the patella and of the olecranon safe procedures. It has made available skeletal traction—the Steinman pin and the ice-tongs. It has shortened the period of disability and incidence of chronic osteomyelitis. Along with asepsis should be noted advantages of the use of tetanus antitoxin.

By specialization, I mean the study of individual fractures.

General surgeons have developed the treatment of fractures: care is what is needed in the treatment of fractures and not a fracture specialist. To return to the individual fracture study: Speed's work has shown a way to reduce the disabilities in fractures of the carpal scaphoid; Ashhurst's study on fractures at the elbow, which won the Gross prize in 1910, has resulted in a treatment which prevents crippling deformities; Whitman's abduction cast has reduced the mortality and increased the number of cases of bony union in fracture of the neck of the femur; Delbet's method of treatment of ankle fractures (introduced into this country by Ashhurst) bids fair to reduce the disability in these cases; subastragalar arthrodesis, advocated by Wilson for fracture of the os calcis, is a method that will rehabilitate a cripple.

The propaganda in the form of fracture meetings, instituted by Scudder, Darrach, Walker, Ashhurst and others, has disseminated the knowledge of the necessity of efficient means of fracture treatment. Their battle-cry is "splint 'em where they lie" and "immediate reduction."

Last, there is the statistical study—tedious work and unproductive of glory, but essential to improvement of methods. Study of end-results has shown to us that we are still lacking an efficient method of treatment in fractures of the hip. Also, it is probably a factor in the search for immobilization methods which will give minimum periods of disability. At any rate, it will afford surgeons seventy-five years hence a means to judge whether we have done as well with our equipment as Hamilton, Gant, Gibson, Velpeau and Barton did with theirs.

CLASSIFICATION AND MECHANISM OF FRACTURES OF THE LEG BONES INVOLVING THE ANKLE

BASED ON A STUDY OF THREE HUNDRED CASES FROM
THE EPISCOPAL HOSPITAL*

By ASTLEY P. C. ASHHURST, M.D.

AND

RALPH S. BROMER, M.D.

SURGEON AND ROENTGENOLOGIST, RESPECTIVELY, TO THE HOSPITAL

ANATOMIC AND SURGICAL STUDY

By DR. ASHHURST

POTT, Dupuytren, Cooper, Maisonneuve, Tillaux, Hönigsmied, Stimson, Destot, Chaput, Quénu—and shall I not add Scudder, Cotton, Roberts and Speed? What can any one say more, at this late day? And yet the fact remains that there is no entirely satisfactory classification of ankle fractures in existence, and that many points of the mechanism of their production still are in dispute. I say there is no entirely satisfactory classification of these fractures in existence, because that which is the best—being the most scientific and complete, namely, that of Quénu—is less a classification than a catalog; and because his strict adherence to an anatomic-pathologic classification and his stern rejection of the historical pathogenetic classification compel him to place side by side lesions in no sense related, except that the fracture lines happen to be similar, and to separate widely other lesions which, though presenting very dissimilar lines of fracture, nevertheless represent only different degrees or stages, or merely variants, of one and the same lesion.

It is perhaps unnecessary to argue the desirability of classi-

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fication, for without classification the relation of one lesion to another can be neither remembered nor understood in any department of knowledge, and comprehension is a prerequisite for intelligent memory and for rational diagnosis and treatment.

HISTORICAL

Si chirurgien expérimenté que vous soyez, ne sautez pas ces premières pages. Elles sont indispensables, mais fatigantes à lire; mieux vaut remettre à un autre jour cette besogne ardue que de l'entreprendre mal disposé.—Farabeuf.

Pott¹ (1769) described a fracture which does not exist, and Dupuytren² (1819) commended him for his acute observation and fidelity to nature. Cooper³ (1822), more sensible than either, merely recounted what he had seen, avoided speculation about what he had not seen, and was silent on subjects about which he had no knowledge. Maisonneuve⁴ (1840) and Tillaux⁵ (1872) studied the mechanism by experiments on the cadaver and, though they were both correct, they came to different conclusions; and Tillaux thought his work had entirely invalidated that of Maisonneuve. Hönigschmied's⁶ (1877) experiments on cadavers (125 in all) may be said to have carried this means of investigation to its limit; and though subsequent students, including myself, have repeated such experiments, it is evident that this method of investigation has many limitations, as it is impossible to reproduce in the cadaver the falls which living patients suffer and the muscular tension to which their limbs are constantly subjected.

Stimson's⁷ study, in 1892, was the last one of importance before the advent of the roentgen-ray; and it was Destot⁸ (1911) who first of all published extensive observations of ankle fractures illustrated by, and based on, roentgenographic studies. It is strange that no English writer since Cooper has made a special study of fractures at the ankle, and that with the exception of Stimson, already mentioned, no American surgeon has made a particular study of the subject, though Scudder, Cotton, Roberts and Kelly, and Speed have discussed it at greater or less length in their textbooks on fractures. It is evident, however, to any attentive student of the subject

that most of the recent writers, if not, indeed, all, demonstrate by their statements that they have not themselves read, or at any rate have not read with understanding, the works of their predecessors—especially of Dupuytren, Cooper, Maisonneuve and Tillaux. Most surgeons think they know what they mean by a “Pott’s fracture,” but, as I said before, there is little use in knowing much or anything about a type of fracture which does not exist.

What is Pott’s fracture? Let Mr. Percivall Pott tell in his own words (1769).⁹

“The limb most commonly preserves its figure and length . . . if the fibula only be broken, in all that part of it which is superior to letter *A* in the annexed figure” (Fig. 1), “or in any part of it between its upper extremity, and within 2 or 3 inches of its lower one. . . . I have already said . . . that the support of the body and the due and proper use . . . of the joint of the ankle depend almost entirely on the perpendicular bearing of the tibia upon the astragalus, and on its firm connection with the fibula. If either of these be perverted or prevented, so that the former bone is forced from its just and perpendicular position on the astragalus, or if it be separated by violence from its connection with the latter, the joint of the ankle will suffer a partial dislocation internally; which partial dislocation cannot happen without not only a considerable extension or perhaps laceration of the bursal ligament of the joint, which is lax and weak, but a laceration of those strong tendinous ligaments which connect the lower end of the tibia with the astragalus and os calcis, and which constitute in great measure the ligamentous strength of the joint of the ankle.

“This is the case when, by leaping or jumping, the fibula breaks in the weak part already mentioned, that is, within 2 or 3 inches of its lower extremity. When this happens, the inferior fractured end of the fibula falls inward, toward the tibia, that extremity of the bone which forms the outer ankle is turned somewhat outward and upward, and the tibia, having lost its proper support and not being of itself capable of steadily preserving its true perpendicular bearing, is forced off from the astragalus inward [Evidently, he means by the weight of the body, after primary fracture of the fibula produced by the first impact of the foot with the ground.] by which means, weak bursal, or common ligament of the joint is violently stretched, if not torn, and the strong ones, which fasten the tibia to the astragalus and os calcis, are always lacerated, thus producing at the

same time a perfect fracture and a partial dislocation, to which is sometimes added a wound in the integuments, made by the bone at the inner angle. By this means, and indeed as a necessary consequence, all the tendons which pass behind or under, or are attached to the extremities of the tibia and fibula, or os calcis, have their natural direction and disposition so altered, that instead of performing their appointed actions, they all contribute to the distortion of the foot, and that by turning it outward and upward." (Note that there is not a word of posterior displacement, and that this is not shown in the plate, here reproduced as Fig. 1.)

This description, and the accompanying illustration, call for several remarks. It is to be noted, first, that Pott's fracture, as described and pictured by himself, is a primary, nearly transverse, fracture of the fibula, attended by a subsequently produced "partial dislocation" of the ankle-joint internally. Second, the fracture of the fibula occurs at, or below, the point marked *A* in the illustration ("within 2 or 3 inches of its lower extremity"); and the upper end of the lower fragment is described as falling in against the tibia, while the external malleolus is turned outward. Third, there then is supposed to occur rupture of the ligaments below the internal malleolus, with a partial dislocation of the tibia inward, off the trochlea of the astragalus, as the latter bone turns outward, around a more or less anteroposterior axis. Now, such a fracture of the fibula does not occur as a type, as may be readily verified by the examination of any series of roentgenograms or postmortem specimens; and if it did occur, it would be impossible for the upper end of the lower fragment to fall in against the tibia, for the reason that the fibula is already closely applied to the tibia at the point described. There is barely room to insert the blade of a scalpel between the bones at a point 3 inches (7.5 cm.), or less, above the tip of the fibula.

It was the particular merit of Dupuytren to systematize the teaching of various pioneers, such as Pott (1769), Bazille¹⁰ (1771), Bromfeild¹¹ (1773) and Pouteau¹² (1783), who had recognized, as fractures, lesions which had been regarded by former generations as dislocations; and it is to Dupuytren's memoir on "The Fracture of the Lower End of the Fibula"¹³

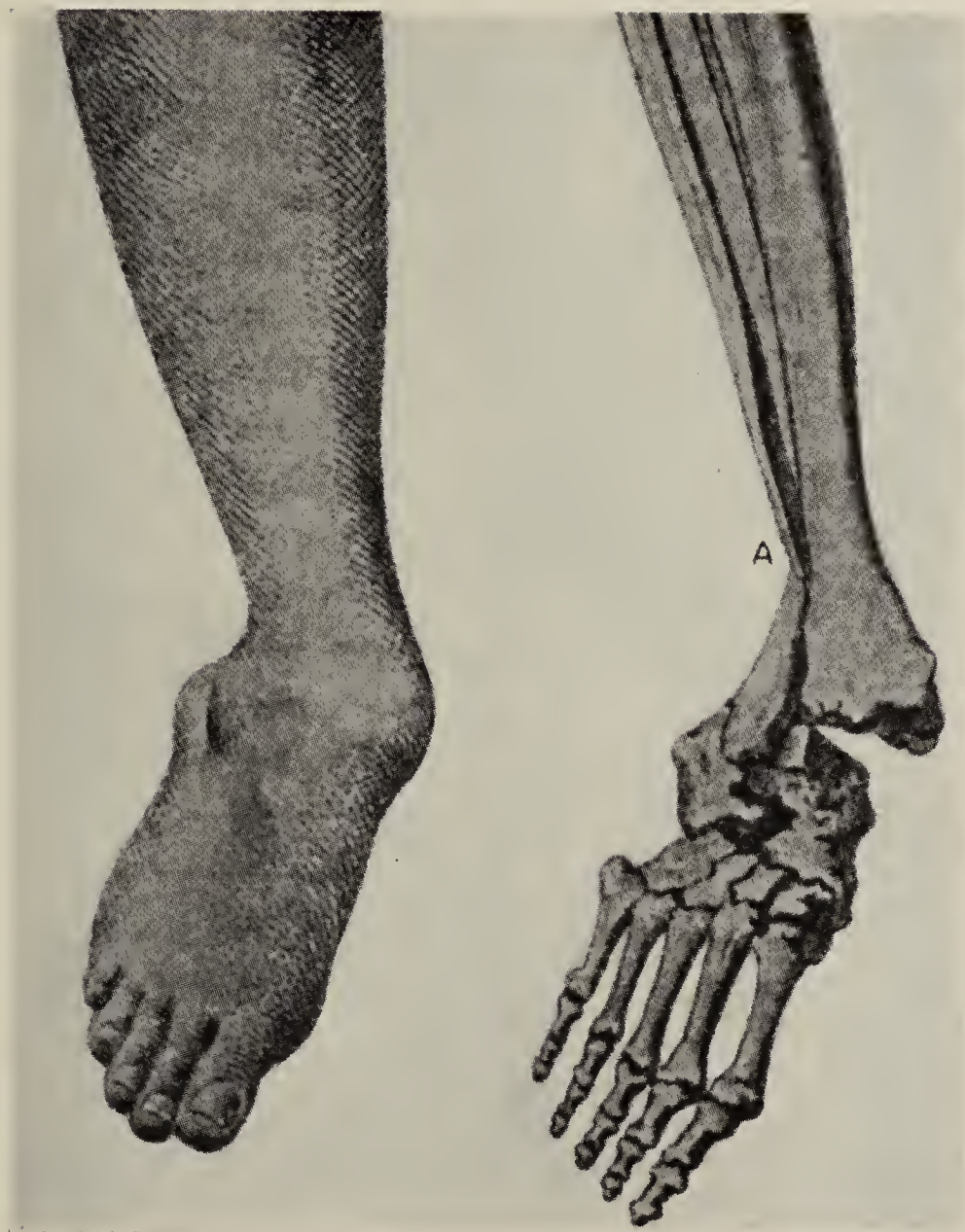


FIG. 1.—Illustration from Pott's work, *Some Few General Remarks on Fractures and Dislocations*, London, 1769, facing p. 69.

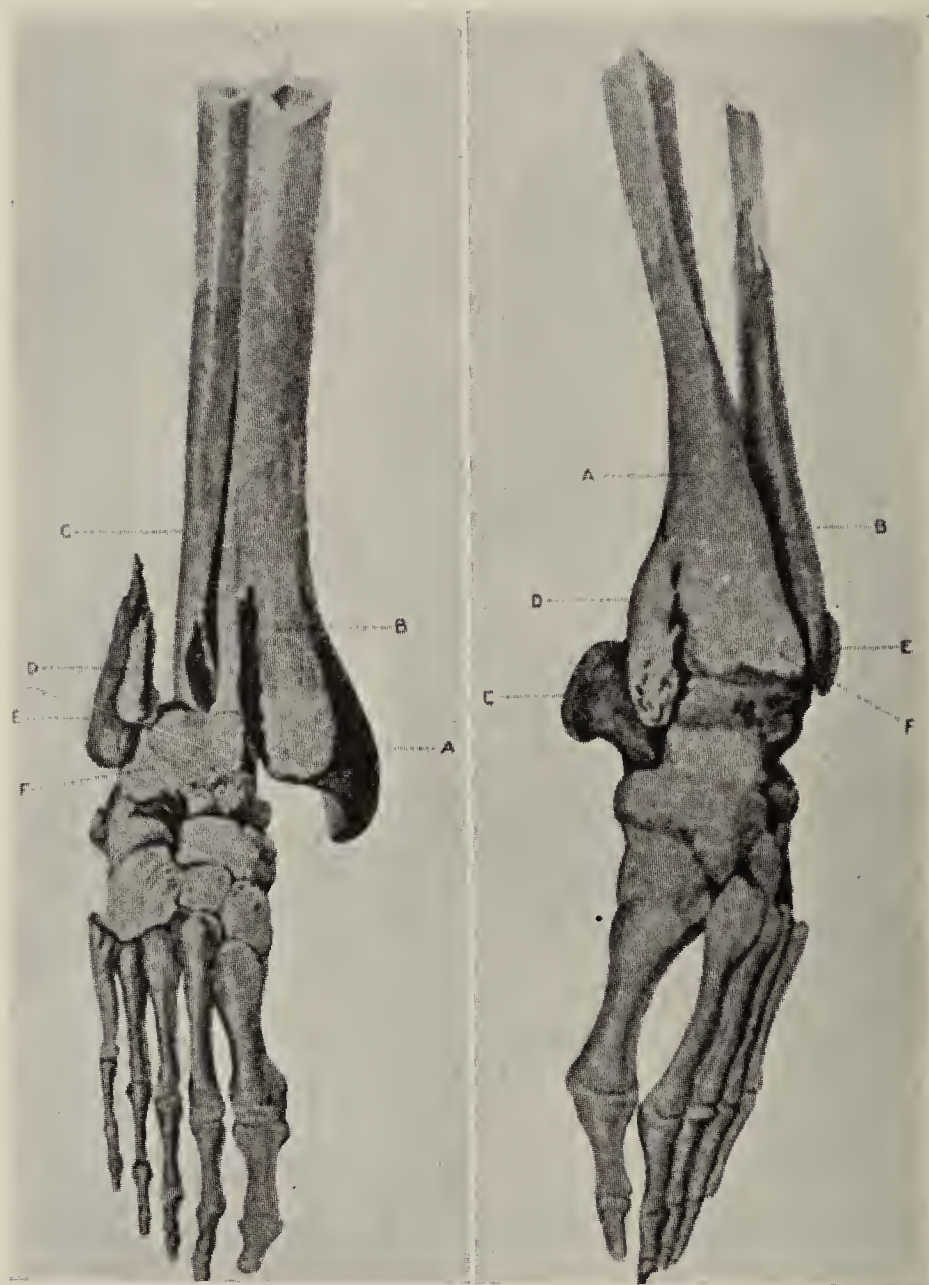


FIG. 2.—Illustrations from Cooper's *Treatise on Dislocations and Fractures of the Joints*, London, 1822, Plate XVI, Figs. 1 and 2. "Fig. 1 shows the dislocation of the tibia inwards at the ankle-joint: *A*, the malleolus internus of the tibia thrown on the inner side of the astragalus; *B*, a portion of the tibia split off; *C*, fibula broken; *D*, the broken portion of the tibia adhering by ligament to the fibula; *E*, the malleolus externus of the fibula, with the broken portion of the tibia adhering to it, and *F*, astragalus thrown outward. Fig. 2 shows the dislocation of the tibia outward at the ankle-joint: *A*, the tibia; *B*, the fibula; *C*, the os calcis; *D*, fracture of the tibia at the malleolus internus, which has become reunited; *E*, extremity of the fibula broken, and *F*, tibia thrown on the outer side of the articular surface of the astragalus, to which it is ankylosed."



FIG. 3.—Cooper's Plate XVII, Figs. 1 and 2: "Partial dislocation of the tibia forwards, at the ankle-joint: Fig. 1: *A*, the tibia thrown forward over the os naviculare; *B*, the astragalus; *C*, new articular surface of the tibia; *D*, the portion of the astragalus behind the tibia. Fig. 2, opposite view of Fig. 1: *A*, the tibia thrown forwards; *B*, new articular surface of the tibia; *C*, astragalus; *D*, fibula broken and reunited; *E*, malleolus externus of the fibula; *F*, astragalus behind the tibia."



FIG. 4.—Transverse section of leg through inferior tibiofibular joint, showing fibula lodged in its groove formed by the anterior and posterior tubercles of the tibia. Note the obliquity of the intermalleolar axis, forming an angle of 30 degrees with the axis of motion of the ankle-joint, which is nearly transverse. Note that the anterior tubercle projects laterally much farther than the posterior, so that in anteroposterior roentgenograms its shadow much overlaps that of the fibula. (From a preparation in the laboratory of operative surgery, University of Pennsylvania.)



FIG. 5.—Posterior view of the ankle-joint, all structures removed except the bandlike ligaments. Note the interosseous membrane, its fibers passing downward from the tibia to the fibula, the similarly directed fibers of the posterior-inferior tibiofibular ligament; the middle band of the external lateral (fibulo-calcanean) ligament; the posterior band of the external lateral ligament (fibulo-astragalar ligament) attached to the lateral tubercle of the astragalus; the posterior fibers of the internal lateral ligament, and the posterior surface of the internal malleolus grooved for the tendon of the tibialis posticus. (From a preparation in the laboratory of operative surgery, University of Pennsylvania.)



FIG. 6.—Frontal section of the ankle-joint. Adduction causes tension on external lateral ligament, and if forced will tear off the external malleolus



FIG. 7.—Frontal section of the ankle-joint. Abduction causes tension on internal lateral ligament, and will rupture this or fracture the internal malleolus before outward pressure on the external malleolus will fracture the latter or cause diastasis between tibia and fibula. (This specimen and that shown in Fig. 6 are from a preparation in the laboratory of operative surgery, University of Pennsylvania.)



that surgery is principally indebted for its understanding of all these ankle fractures. It is true that Gosselin (1872) reproached Dupuytren with having propounded his theories of mechanism merely by means of his reasoning, and not on a basis of clinical observations or cadaveric experiments (though Dupuytren made a number of the latter, and presented records of 207 patients with ankle fractures). But there is much justification for Dupuytren's¹⁴ statement that the mechanism detailed by patients cannot be relied on, since it is well known that in dislocations of the shoulder, for instance, they all will say the injury resulted from a fall on the point of the shoulder (because that is where they feel the pain), whereas the state of their elbow or hand proves quite the contrary. Similarly, I have known a patient with an ankle fracture badly united in abduction (fibular flexion) to assert that it was produced by adduction (tibial flexion), because her heel was prominent beneath the internal malleolus just after the accident; not realizing that the heel had been brought into that position by outward displacement of the point of the foot, the astragalus turning around the long axis of the leg.

Now it is to be noted that Dupuytren, who commended Pott for his accurate delineation of the typical fracture of the lower end of the fibula, was evidently of the belief that the typical fracture which he himself was describing was the same as that of Pott; and that this is still the opinion of the French is manifest from a footnote of Quénu,¹⁵ in which he says that the French mean by Dupuytren's fracture precisely what the English mean by Pott's. So that it is somewhat confusing to have J. Hutchinson, Jr.¹⁶ and some recent writers name and illustrate as Dupuytren's fracture a lesion which Dupuytren¹⁷ encountered only once in more than 200 cases, and which consisted in a fracture of the fibula, rupture of the tibiofibular ligaments, and displacement upward of the astragalus along the fibular side of the tibia. As it is a fact that the fracture at the ankle most often seen, and therefore the most typical fracture, is that first described accurately by Maisonneuve (1840),¹⁸ it is safe to assume that both Pott's and Dupuytren's descriptions applied to this fracture and not to the rather hypothetical type they thought they were describing.

However, there is a typical, though rare, fracture at the ankle which corresponds closely enough to the original illustration of Pott to make it worthy of being called by his name; it is a typical "flexion fracture" (Biegungsbruch) of the fibula, usually 8 cm. ($3\frac{1}{4}$ inches) or *higher* above the tip of the external malleolus, accompanied by fracture of the internal malleolus and almost invariably by rupture of the inferior tibiofibular ligaments, permitting a diastasis between tibia and fibula. It is such a fracture as is illustrated in Fig. 26; and may well be called Pott's fracture, even though Pott in his own description placed the seat of the fibular fracture too low, and ignored the fracture of the internal malleolus and the tibiofibular diastasis.

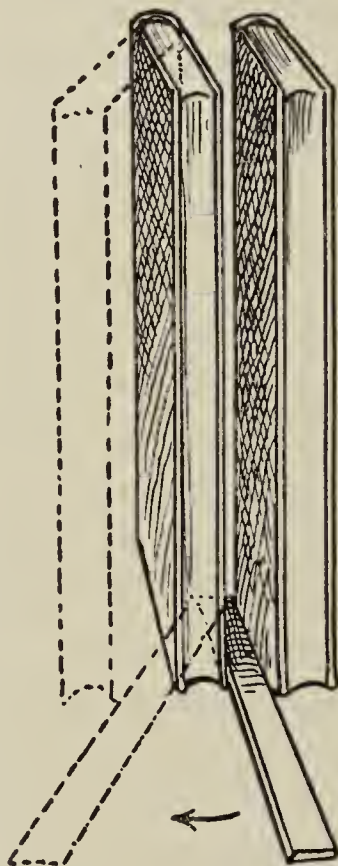


FIG. 8.—An illustration of Maisonneuve's explanation of fracture of the external malleolus by outward rotation of the foot around the axis of the leg. The books represent the malleoli and the ruler represents the foot. (See page 274.)

Cooper³ (1822) did not get away from the idea of the paramount importance of the dislocation of the tibia in these lesions (nor, it may be remarked, did Malgaigne, Hamilton, Treves or Stimson, many years later); and he described the fractures as mere incidents. But any modern surgeon will

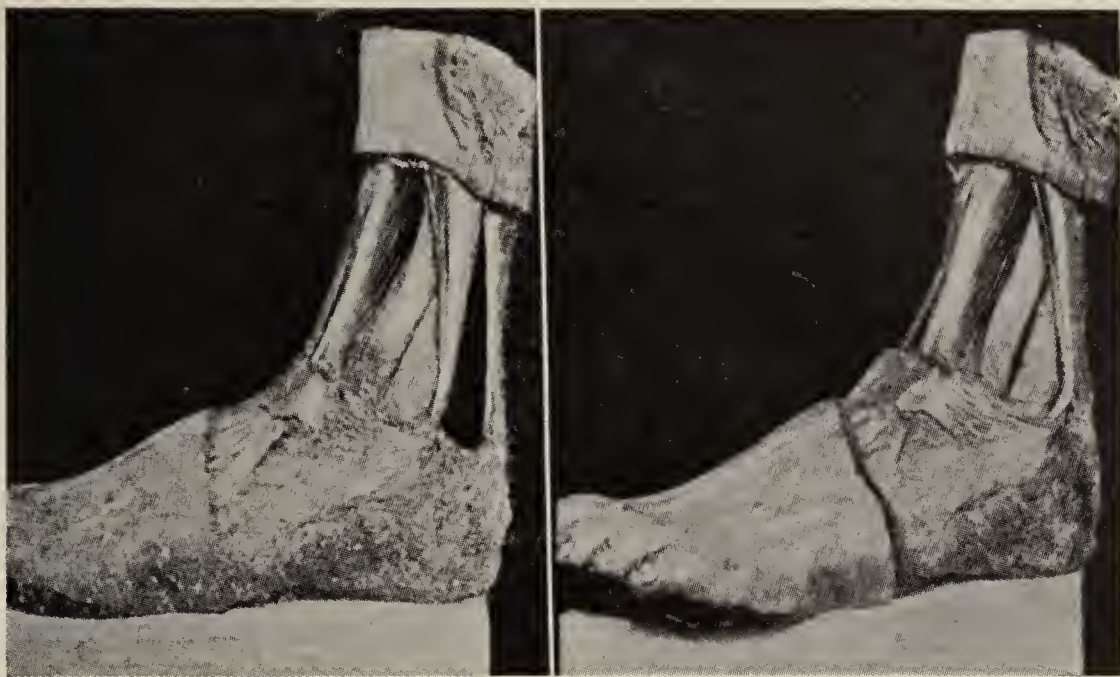


FIG. 9.—Oblique fracture of the fibula through the inferior tibiofibular joint, produced by an osteotome: In the first figure the foot is in the anatomic position; in the second it has been rotated outward around the axis of the leg, which is the only motion that will cause separation of the fragments. (See page 306.)



FIG. 10.—Mixed oblique fracture of the lower end of the fibula: The first lesion resulting from external rotation (A, I); occurs as isolated lesion in 25 per cent of all cases of fracture of the ankle; as a combined lesion in 61 per cent. Usually invisible in anteroposterior views.



find it difficult to bring forth as extensive a list of severe ankle lesions as that recorded by Cooper, though he can easily excel Cooper in the number of lesions which (owing to the absence of deformity) can be certainly recognized only with the aid of the roentgen-ray.

Cooper describes succinctly, but accurately, the following main groups of lesions; and scarcely one additional type has been discovered since, even with the aid of roentgenography:

1. *Simple Dislocation of the Tibia Inward.* In this case there is a fracture of the fibula, 2 inches above its tip, carrying with it an attached fragment of the tibia;¹⁹ the lower end of the upper fragment of the fibula rests on top of the astragalus,²⁰ and the tibia with the internal malleolus intact descends on the median surface of the astragalus. (This is illustrated in Fig. 1 of Plate 16 of Cooper's monograph, first edition, 1822, and is reproduced here as Fig. 2. The figure was copied by Vidal de Cassis²¹ in his second edition (1846) as Fig. 53, and the lesion was for many years known in France by his name until Quénu recently called attention to the fact, quite clearly stated by Vidal, that the illustration was copied from Cooper.) This, says Cooper, is the most frequent of the dislocations of the ankle. It corresponds probably to the form now known to the French as the "low Dupuytren" fracture, though in the latter type, which is very frequent, there is very seldom any intermediate fragment detached from the tibia.

2. *Simple Dislocation of the Tibia Forward.* In this case there is a fracture of the fibula, 3 inches above its tip; the internal lateral ligament is partly lacerated; the tibia and upper fragment of the fibula advance forward, and the tibia rests on the upper surface of the scaphoid and internal cuneiform. In *partial dislocation of the tibia forward*, the articular surface of the tibia is divided in two, the anterior part rests on the scaphoid and the posterior on the astragalus. The fracture of the fibula (as shown in Plate 17, Figs. 1 and 2, of Cooper's monograph, reproduced here as Fig. 3) runs obliquely up and back, through the inferior tibiofibular joint. Thus there is here accurately described the posterior marginal fragment of the tibia, which many recent writers think they were themselves the first to discover after the introduction of roentgenography (Destot, Chaput, Cotton, Sear, etc.).

3. *Simple Dislocation of the Tibia Outward.* This, says Cooper, is the most dangerous of the three, for it is produced by greater violence, etc. The internal malleolus is obliquely fractured and

separated from the shaft of the bone; the fractured portion sometimes consists only of the malleolus; at other times, the fracture passes obliquely through the articular surface of the tibia, which is thrown forward and outward on the astragalus before the external



FIG. 11.—Mixed oblique fracture of the fibula, with great obliquity. In these fractures the obliquity may be great or slight; but the line of fracture is always higher on the posterior than on the anterior border of the fibula and in 84 per cent of the cases the anterior end of the fracture line is between the tip of the malleolus below and the level of the articular surface of the tibia above. In 8 per cent of the cases the anterior end of the fracture line passes through the fibula at the level of the anterior tubercle of the tibia, and in 8 per cent its anterior end is above this level (in which circumstances the tubercle is detached or there is diastasis). All tracings here reproduced were made directly from the roentgenograms.

malleolus. The astragalus is sometimes fractured, and the lower extremity of the fibula is broken into several splinters. The internal and external lateral ligaments are usually intact; but if the fibula is not broken, the external lateral ligaments are ruptured. (Note that there is here described the “fracture by adduction,”

with a splitting-off of a greater or less portion of the medial surface of the tibia—illustrated in Fig. 2, from Cooper—a lesion which Tillaux thought had not before his time been observed by any surgeon.)



FIG. 12.—Mixed oblique fracture of fibula, with fracture of internal malleolus (A, II, b). The fibular fracture is visible only in the anteroposterior view (rare). Note that there is considerable lateral displacement of the astragalus, but that the posterior displacement is only apparent, not real, being due to the external rotation of the foot.

Under the heading “Fractures of the Tibia and Fibula Near the Ankle-joint” (p. 353), Cooper describes a fracture of the fibula from 2 to 3 inches above the ankle-joint (that is, not at a point from 2 to 3 inches above the tip of the external malleolus, but at the point where occurs the fracture called by Pott’s name), produced by falling laterally while the foot is confined in a deep cleft; and a fracture of the tibia, in which the fracture line runs either obliquely down and in, toward the internal malleolus (*i. e.*, the ordinary spiral fracture of the shaft low down), or obliquely from 1 or 2 inches above the internal malleolus down and out into the ankle-joint (*i. e.*, the ordinary splitting fracture of the median part of the articular surface, usually dependent on a primary fracture of the fibula, and forming a less-advanced degree of the fracture by

adduction, already described by Cooper as an outward dislocation of the tibia).

After Cooper, Maisonneuve⁴ (1840). In a most remarkable essay, which has been ignored by most subsequent writers, Maisonneuve threw more light on the subject of ankle fractures than has any one since.

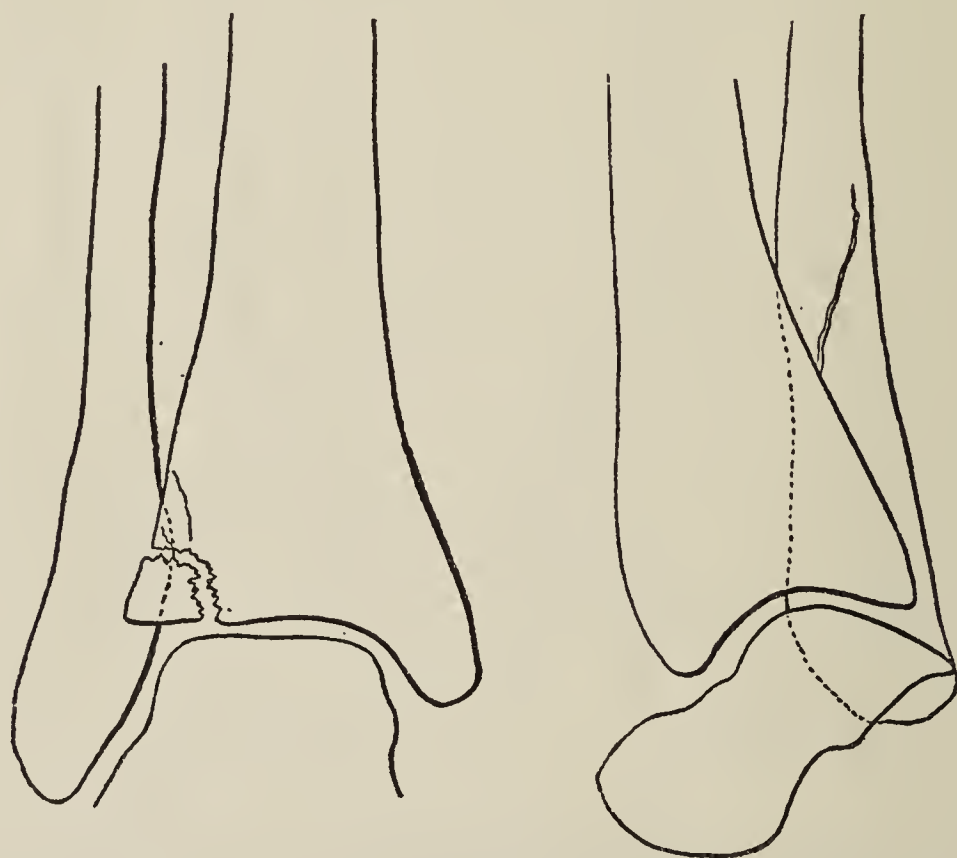


FIG. 13.—Mixed oblique (incomplete) fracture of fibula, complicated by, and subsequent to, avulsion of the anterior tubercle of tibia. (See p. 308.)

Up to that time, as he says, two theories prevailed to explain the mechanism of fractures of the lower end of the fibula: (1) by adduction, by which means the tip of the external malleolus was torn off; this was thought by Dupuytren to be the most frequent variety,²² and (2) by abduction, when, from pressure upward and laterally by the astragalus on the external malleolus, the latter was forced outward until the fibula broke at its weakest point, namely, entirely above the malleolus, that is, in the region described by Pott; this fracture being usually accompanied by a secondarily produced fracture of the internal malleolus or rupture of the internal lateral ligament. The second variety was thought by everybody, except Dupuytren, to be the most frequent.

Now Maisonneuve proposed another mechanism, which he believed explained the production of the most common type of

fracture: this was simple deviation of the point of the foot outward, that is, external rotation of the foot in the tibiofibular mortise around a vertical axis. In one recent necropsy he had found an oblique fracture of the fibula, the line of fracture beginning on the anterior surface of the external malleolus 4 cm. above its tip and extending upward and backward to a point on the posterior surface of the fibula 8 cm. above the tip of the external malleolus. And he found that precisely this fracture was readily produced in the cadaver by the mechanism of outward rotation, as above mentioned, whenever the tibiofibular ligaments held firm.

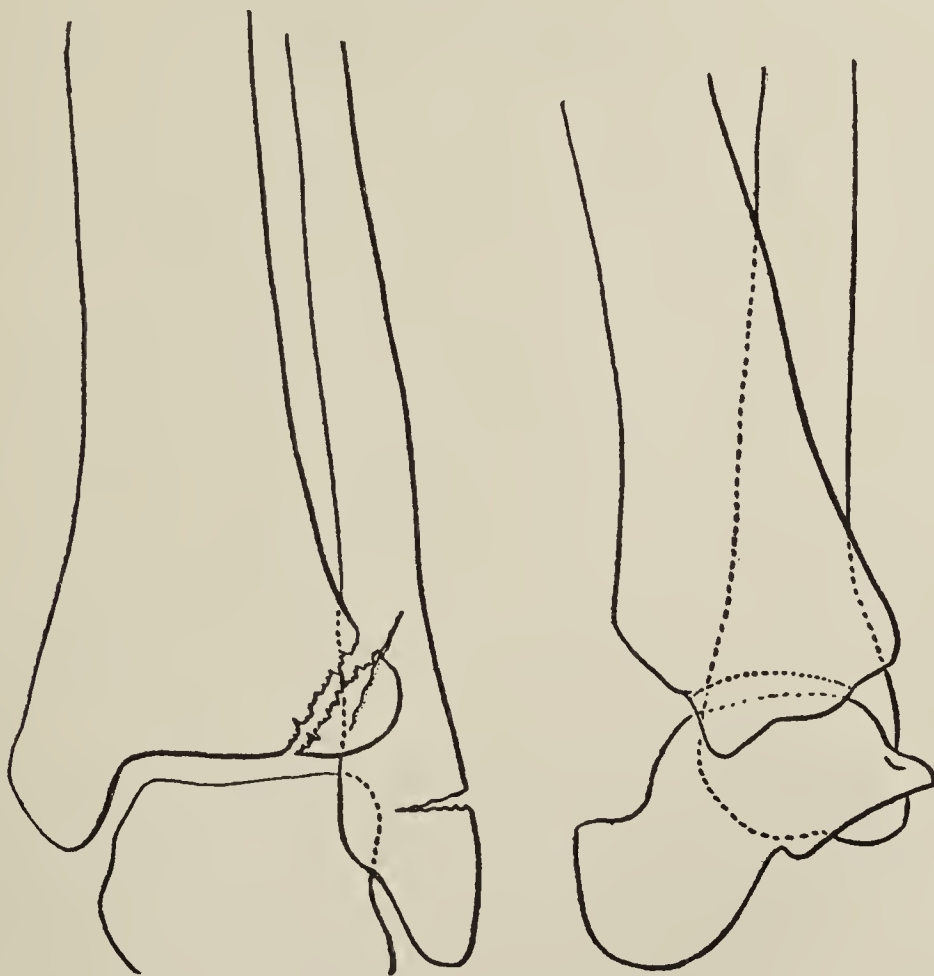


FIG. 14.—Mixed oblique (incomplete) fracture of fibula complicated by, and subsequent to, avulsion of anterior tubercle of tibia.

So far as I can ascertain, Maisonneuve was the first and, I might say, almost the only writer up to the present day to appreciate properly the importance of the inferior tibiofibular ligaments in the mechanism and classification of ankle fractures; and to recognize the great frequency of the oblique fracture of the fibula. This fracture, he found, was always the first lesion following external rotation of the foot, and occurred without any ligamentous injury. If the external rotation of the foot

were continued far enough, the internal lateral ligament would rupture, or often the internal malleolus would be pulled off. The resulting deformity, he found, was precisely the picture drawn by Dupuytren ("*coup de hache*," etc.); and all of these phenomena disappeared when the position of the foot was corrected. If the inferior tibiofibular ligaments break during this external rotation of the foot around the long axis



FIG. 15.—Incomplete fracture of fibula below its head (A, I, variant), apparently from compression in its long axis (see pages 309 and 313). No bone lesion at ankle. There was a history of a fall down three steps while carrying a load of 100 kilograms on the back. Injury occurred so quickly the patient did not know whether he turned his ankle or struck the side of his leg against the marble steps. Pain was so great that he had to be brought to the hospital by a patrol wagon. Examination showed fracture in the upper third of the left fibula (crepitus and deformity). The ankle was swollen over the external malleolus.

of the leg, there occurs a greater or less diastasis of the inferior tibiofibular joint; and if the movement still continues, the fibula breaks not at the usual site, but in its upper third, or at least in its middle third. It is to this fracture of the fibula in its upper third, produced after diastasis of the inferior tibiofibular joint has occurred, that the name of Maisonneuve's fracture has been attached, though he never encountered such a case clinically.

Maisonneuve's chief contribution was his recognition of the oblique fracture of the lower end of the fibula as the first stage of a lesion frequently including also a fracture of the internal malleolus or a rupture of the internal lateral ligament; and it is a pity that his name is not applied to this very frequent fracture rather than to one of the utmost rarity. Moreover,

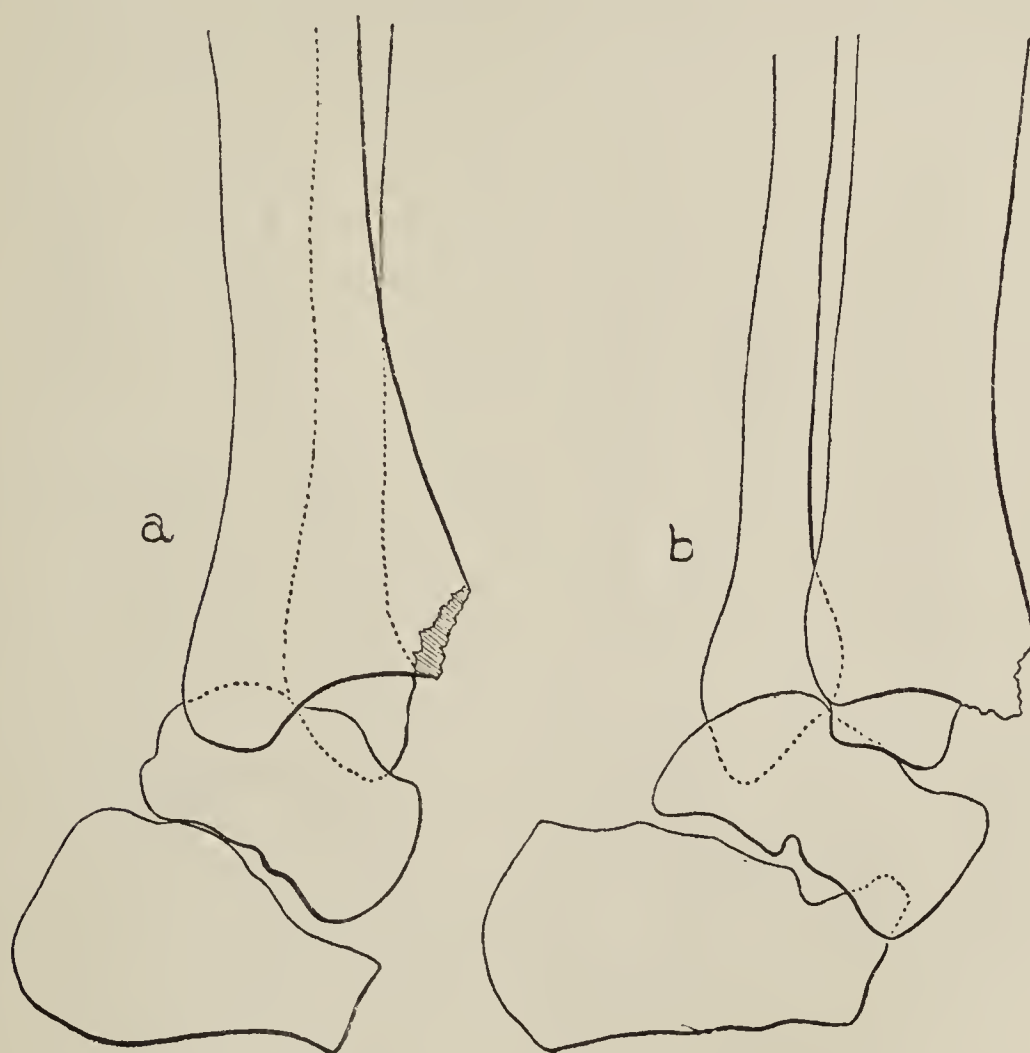


FIG. 16.—Diastasis with displacement of fibula behind tibia. The anterior tubercle of tibia has been torn off and accompanies the fibula, which is not fractured. Diastasis by external rotation (mechanism described by Huguier⁵⁰): (a) anteroposterior view, and (b) lateral view before reduction. (See also Fig. 17 (A, I, complication), and page 309.)

he was no doubt correct, as I have previously intimated, in believing that this was the lesion whose clinical signs were described by Dupuytren, and that the latter erroneously thought these cases were such as had been depicted by Pott. Maisonneuve's chief and almost his only error, as it seems to me, was his failure to appreciate that a lesion did exist, consisting of a fracture well above the inferior tibiofibular joint,

associated with fracture of the internal malleolus or rupture of the internal lateral ligament, and that this type of fracture, though rare, was yet much less rare than a fracture of the fibula in its upper third or even in its middle third.

After Maisonneuve, Tillaux⁵ (1872). Tillaux did not distinguish in his cadaveric experiments between abduction (fibular flexion) of the foot and the same movement combined with slight outward rotation (*i. e.*, around the long axis of the

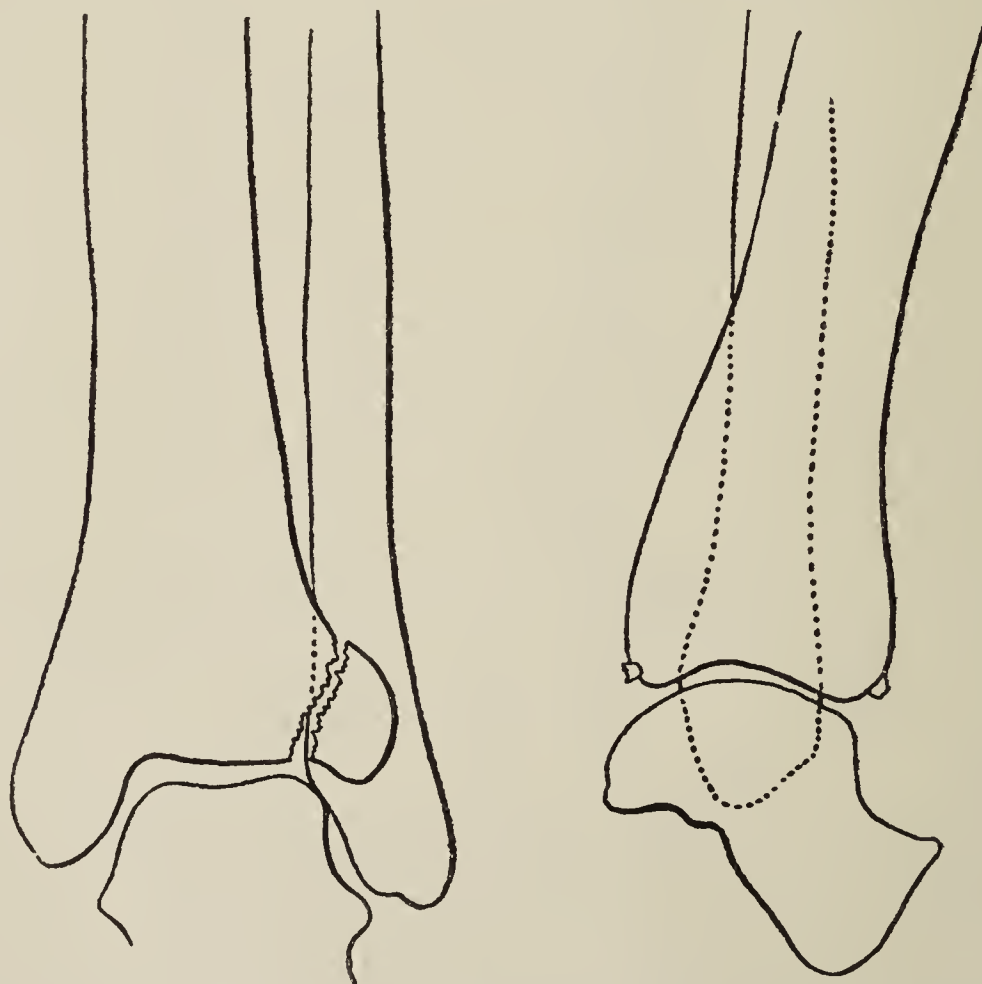


FIG. 17.—Same case as that shown in Fig. 16, after reduction. Fracture of anterior tubercle of tibia clearly seen.

leg). Abduction, he found, caused (*a*) rupture of the internal lateral ligament or tearing off of the internal malleolus, or (*b*) the same lesions plus fracture of the fibula above the inferior tibiofibular ligaments, or 6 to 7 cm. above its tip; the tibiofibular ligaments may not rupture, but the degree of rupture of these ligaments determined the degree of the displacement; and he found the fibula could not be broken in abduction movements unless the internal malleolus or the internal lateral ligament had been previously broken. He also called

particular attention to the great frequency of a fragment of bone torn off the lateral border of the tibia by the inferior tibiofibular ligaments; this fragment has since been known to the French as the "third fragment of Tillaux" (remember it had been described and illustrated by Cooper and by Vidal de Cassis).²³

It is thus seen that while Maisonneuve minimized the mechanism of pure abduction, Tillaux magnified it out of all reason; for the fact remains that the vast majority of fractures are not of the type produced experimentally by Tillaux by abduction (and which correspond rather to the original type with which Pott and Dupuytren thought they had to deal), but are of the type produced experimentally by Maisonneuve by external rotation. In his description of the results of adduction (tibial flexion), Tillaux agreed with his predecessors and successors (the results are noted below under the account of Hönigschmied's experiments); but he also describes a rare result of adduction, which consists in a transverse supramalleolar fracture of the tibia sometimes occurring in those cases in which the fibula breaks above the inferior tibiofibular joint instead of below it (Fig. 43). This mechanism, he says, involves great strain on the superior tibiofibular joint until the fibula breaks above its malleolus; and he observed a case in life in which this transverse supramalleolar fracture (of the tibia only) was complicated by a diastasis of the *upper* tibiofibular joint. (In this connection, one of the specimens from the Mütter Museum⁷² is of much interest.)

Hönigschmied⁶ (1877), as already remarked, made 125 experiments on the cadaver, to determine the mechanism of ankle fractures, and a brief summary of them is given, as it will be necessary to refer to them frequently in the latter portions of this memoir.

1. *Plantar hyperflexion, twenty experiments.* In 14 cases he got rupture of the internal lateral ligament and anterior fibers of the external lateral ligament; the rupture sometimes occurred from the tarsus, sometimes from the leg bones, sometimes a small fragment of bone was torn loose; in 5 cases (aged subjects), one or both of the malleoli were broken.

2. *Dorsal hyperflexion*, 21 experiments, always with previous division of the tendon of Achilles. In 17 the internal malleolus was broken, by push of the tarsus against its tip. In 2 the internal lateral ligament was ruptured. In 2 the only lesion was in the tarsus.

3. *Tibial flexion (adduction or supination)*, 17 experiments. In all the external malleolus (5) or external lateral ligament (12) was broken. He never got fracture of the internal malleolus or of the tibia.

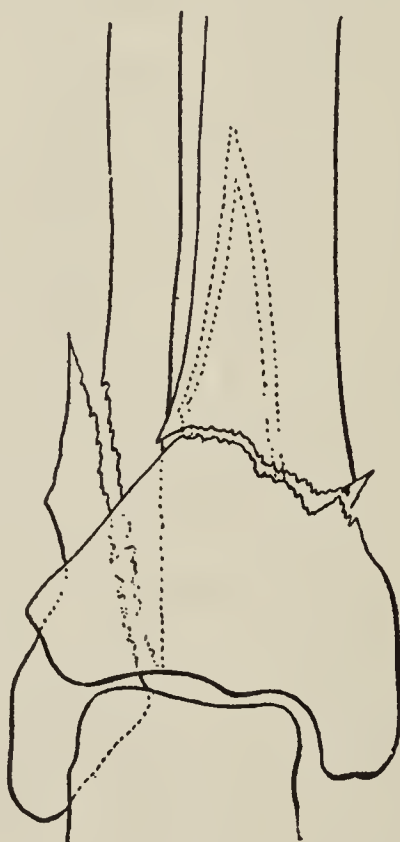


FIG. 18.—Mixed oblique fracture of fibula, internal malleolus represented by whole lower end of tibia (A, III).

4. *Fibular flexion (abduction or pronation)*, 22 experiments. In 15 the internal lateral ligament or the internal malleolus broke. In 7 the only lesions were in the tarsal (2) or subastragalar (5) ligaments. He never obtained a primary fracture of the external malleolus: in 2 cases only did it break, the lesion being a compression fracture at its tip. (If the inferior tibiofibular ligaments had previously ruptured, the weight of the patient's body, in a fall, would have fractured the fibula above these ligaments.)

5. *Inward rotation of foot around long axis of leg*, 20 experiments. In 19 there were lesions of the ligaments (15 involved the tarsal ligaments, of which 6 involved also the external lateral ligament; while in 4 the external lateral ligament was alone involved). In 1 the anterior margin of the external malleolus was broken, evidently

being torn off by the anterior fibers of the external lateral ligament. [Such a fracture had been described by Wagstaffe,²⁴ in 1875, and was later studied by L. LeFort²⁵ (1886) and by his pupil LeRoy²⁶ (1887)].



FIG. 19.—Mixed oblique fracture of fibula, with fracture of internal malleolus and of whole lower end of tibia. There is also a fracture of the anterior tubercle of the tibia (A, III).

6. *Outward rotation of foot around long axis of leg, 22 experiments:*
 (1) In 20, fracture of the lower end of the fibula occurred: (a) in 14 of these there was no diastasis of the inferior tibiofibular joint; in 2 there was rupture of the external lateral ligament with sprain-fracture of the tip of the external malleolus. In 7 there was only slight obliquity of the fracture line, upward and backward (4 had no other lesion, 3 had also rupture of the internal lateral ligament). In 5 the line of fracture was distinctly oblique, involving more of the posterior surface of the fibula (2 of these 5 had also rupture of the internal lateral ligament). (b) In 6 cases there was diastasis,

usually detaching a small intermediate fragment from the tibia, and with rupture of the internal lateral ligament or fracture of the internal malleolus; in 4 of these 6 cases the fracture of the fibula was oblique upward and backward, and in the remaining 2 cases the fibula was broken obliquely in its upper third (*i. e.*, Maisonneuve's fracture). (2) In 2 cases diastasis occurred without fracture of the fibula.

Hönigschmied concluded that these oblique fractures of the lower end of the fibula, produced during external rotation, are not due, as Maisonneuve taught, to the outward pressure of the astragalus on the anterior border of the external malleolus, but to the pull exerted by the posterior band of the external lateral ligament, which tears off the posterior portion of the malleolus.



FIG. 20.—Abduction fracture, first degree (B, I): avulsion of internal malleolus. In this patient the internal malleolus extended abnormally low. This fracture occurs in 6.6 per cent of all fractures at the ankle. All forms of the abduction type together comprise about 21 per cent of the cases.

Destot, in the thesis of Bondet²⁷ (1899) and in his own monograph⁸ (1911), attempted a physiologic classification, abandoning that based on the mechanism. He pointed out that the main function of the tibia was that of support, while the fibula acted merely as a splint along the outer side of the ankle-joint to maintain the direction of the foot. Thus his

classification embraces (1) those fractures which involve only the mortise and disturb the equilibrium of the foot; and (2) those which involve the "pilon tibial" (the tibial "pestle") and hence compromise the support of the body. Quénu²⁸ (1912) addressed very severe criticisms against this classification, and, pointing out that the gravity of the lesions depends entirely on whether they are isolated (involving one bone only) or associated (involving both tibia and fibula), erected

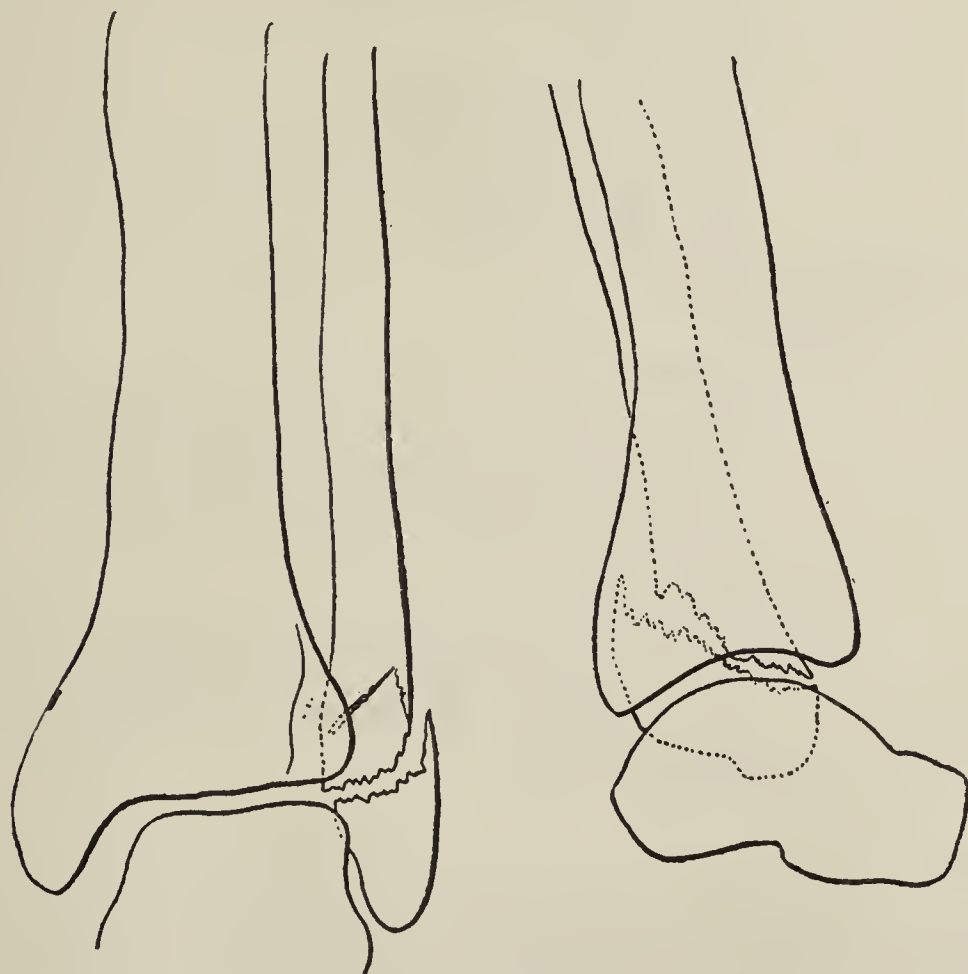


FIG. 21.—Fracture of fibula below inferior tibiofibular ligaments, with very slight obliquity, accompanied by rupture of the internal lateral ligament, as evidenced by lateral displacement of astragalus. This type of fracture is intermediate between the fracture by external rotation already discussed and the fracture by abduction. It is impossible from the roentgenogram alone to say to which of the two types such a fracture really belongs (A, II or B, II).

on this basis an elaborate scheme which, as I have already said, is less a classification than a catalog.

Finally, Tanton²⁹ (1916) attempted to combine the merits of Destot's and of Quénu's classifications, with a reasonable degree of success. Indirect forced movements, he said, may involve (1) the malleoloastragalar or tarsal ligaments (sprain);

(2) the inferior tibiofibular ligaments (diastasis); (3) the malleoli (isolated fractures), very frequent; (4) the "pilon tibial" (isolated), very rare; (5) the malleoli and tibial *pilon* (frequent). Thus he tabulated the lesions as follows (omitting sprains and simple diastases):

I. Fractures of malleoli—

1. Isolated:

- (a) External malleolus.
- (b) Internal malleolus.

2. Associated:

- (a) Low bimalleolar.
- (b) Low Dupuytren.
- (c) Typical Dupuytren (Pott's).
- (d) Maisonneuve.

II. Fractures of pilon—

A. Partial.

1. Isolated:

- (a) Anterior or posterior tubercles of tibia.
- (b) Marginal: anterior, external, posterior.
- (c) Wedge-shaped internal fractures.

2. Associated:

- (a) Anterior margin with a malleolar fracture.
- (b) External margin with a malleolar fracture.
- (c) Posterior margin with a malleolar fracture.

B. Total (Eclatements).

Stimson,⁷ both in his lecture of 1892 and in the last edition³⁰ (1917) of his book on *Fractures and Dislocations*, makes a distinction between fractures in which the mechanism of fibular flexion (abduction) predominates and those in which outward deviation of the point of the foot (outward rotation) predominates.

In the former, he says, the internal malleolus is broken, followed by rupture of the inferior tibiofibular ligaments (or sometimes detachment of a piece of the tibia), and then occurs a fracture of the fibula "close above the malleolus;" while in the latter there occurs rupture, first, of the anterior inferior tibiofibular ligament, then of the anterior fibers of the internal lateral ligament, and almost coincidentally the fibula breaks by the twisting of its lower end, the line of fracture being very oblique.

But, in spite of all this comparatively explicit and positive teaching for so many generations, the fact is undeniable that the average surgeon knows little or nothing about ankle fractures, and that most teachers of surgery either leave their students with but a confused notion of the subject or else teach them positively what is certainly not correct. These examples may be cited:



FIG. 22 (B, II, a).—Fracture of both malleoli by abduction. A heavy steel I-beam, lying on the ground beside the patient while he was standing at work was tipped over and struck the lateral surface of his leg, causing sudden abduction of the foot. He was not knocked down, but he felt a sudden pain in the ankle. He was able to walk on the leg. In the lateral view the fracture of the external malleolus (which occurred subsequent to that of the internal malleolus, and was incomplete and subperiosteal) is not visible at all, while even that of the internal malleolus can hardly be seen.

“Fracture of the lower end of the fibula is a very frequent injury, resulting from indirect violence, the foot, as a rule, being turned violently outward (abduction fracture); as the astragalus forces the external malleolus outward, the tibiofibular ligaments act as a fulcrum, so that the fibula is bent in against the tibia above the attachment of these ligaments, and finally breaks at this point from 5 to 8 cm. above the ankle-joint; the internal malleolus often

is avulsed from the tibia at the same time; and to this combined lesion the name of Pott's fracture is given." (Ashhurst: Surgery, Philadelphia, 1914, p. 380.)

"What is a Pott's fracture? . . . It is a fracture of the lower end of the fibula that is produced by the foot turning outward when the injury is sustained. What happens when the foot turns outward? The external surface of the astragalus presses against the tip of the external malleolus, and the fibula breaks primarily. If the interosseous ligament is strong, the fibula breaks within the first $2\frac{1}{2}$ inches above the tip of the external malleolus, otherwise

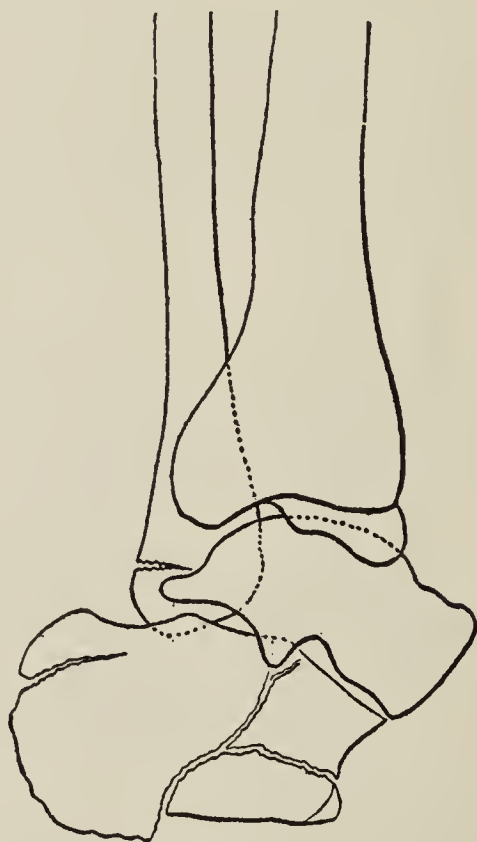


FIG. 23.—Crush fracture of calcaneum permitting fracture of external malleolus by compression. (See page 305.)

only the tip of the fibula is broken off on a line with the tibio-astragaloid articulation, as in the case before us today. In a typical Pott's fracture the fibula fractures higher up.

"Next occurs a fracture of the internal malleolus, due to traction on the internal lateral ligament or rupture of the ligament. Then, as the pressure is continued with the foot everted and the weight of the patient's body is brought to bear on the foot, what happens? The astragalus is crowded up against the tibiofibular articulation, and, acting as a wedge, forces apart the tibia and fibula and splits or tears the interosseous ligament."³¹

"The mechanism of Pott's fracture consists, first, in forcible

eversion of the foot until the outer surface of the astragalus is driven against the fibular malleolus; secondly, in fracture of the shaft of the fibula, usually above the inferior tibiofibular joint, the unyielding interosseous ligament acting as a fulcrum; thirdly, by a continuation of the vulnerating force, the acute superoexternal border of the astragalus develops as a wedge and is driven upward between the tibia and fibula until it springs the joint by lacerating the interosseous ligament."³²

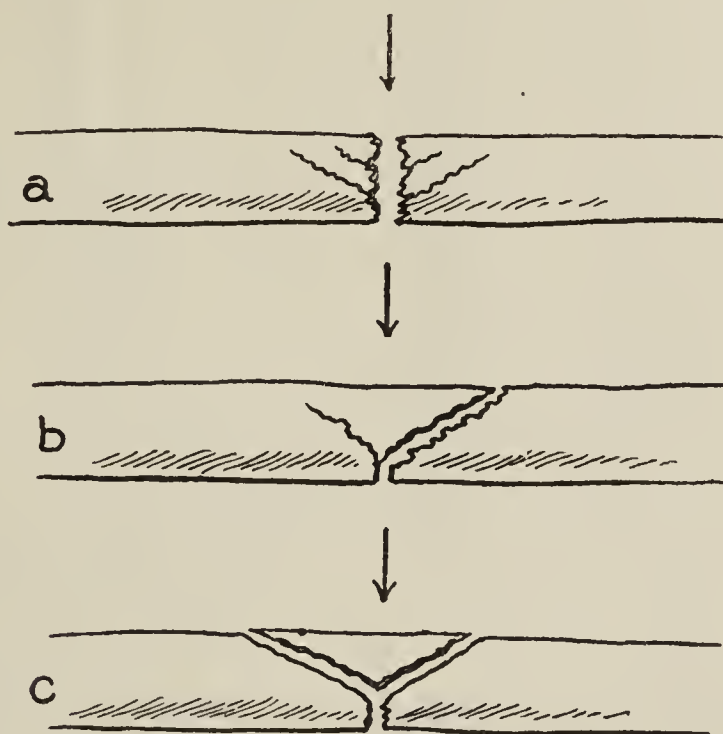


FIG. 24.—Messerer's diagram illustrating the mechanism of fracture by bending (*Biegungsbruch*) or flexion: (a) minute examination always shows in fractures apparently transverse delicate lines diverging from the main fracture and extending backward toward the surface of the bone which has been compressed or flexed, and away from the surface of extension; (b) an oblique fracture represents one of these diverging lines which has become a complete fracture; (c) very frequently a wedge-shaped fragment is detached, with its base on the compression (concave) surface and its apex toward that of extension (convex surface). (See Figs. 25, 26, 28, 29, 35 and 36.)

Cotton³³ (1910) describes all "eversion fractures" as Pott's fractures, and, though he points out the inaccuracy of this term, does not dwell particularly on their mechanism, merely quoting Stimson's account already given.

Roberts and Kelly³⁴ (1916), in describing "fracture by eversion and abduction," say the mechanism is, first, fracture of the internal malleolus or rupture of the internal lateral ligament; "as the force continues the astragalus is forced against the external malleolus, fixing the latter, at the same time the weight of the body falling outward carries with it the leg and the fibula; the fibula is checked

by the astragalus [Do they mean that the external malleolus is kept from moving inward as the body of the fibula moves outward?] and kept attached to the tibia by the tibiofibular ligament. Either the latter ruptures or a line of fracture occurs at the weakest point of the fibula just above the point of attachment of the tibiofibular ligament. It is here that the fixed portion of the fibula meets the

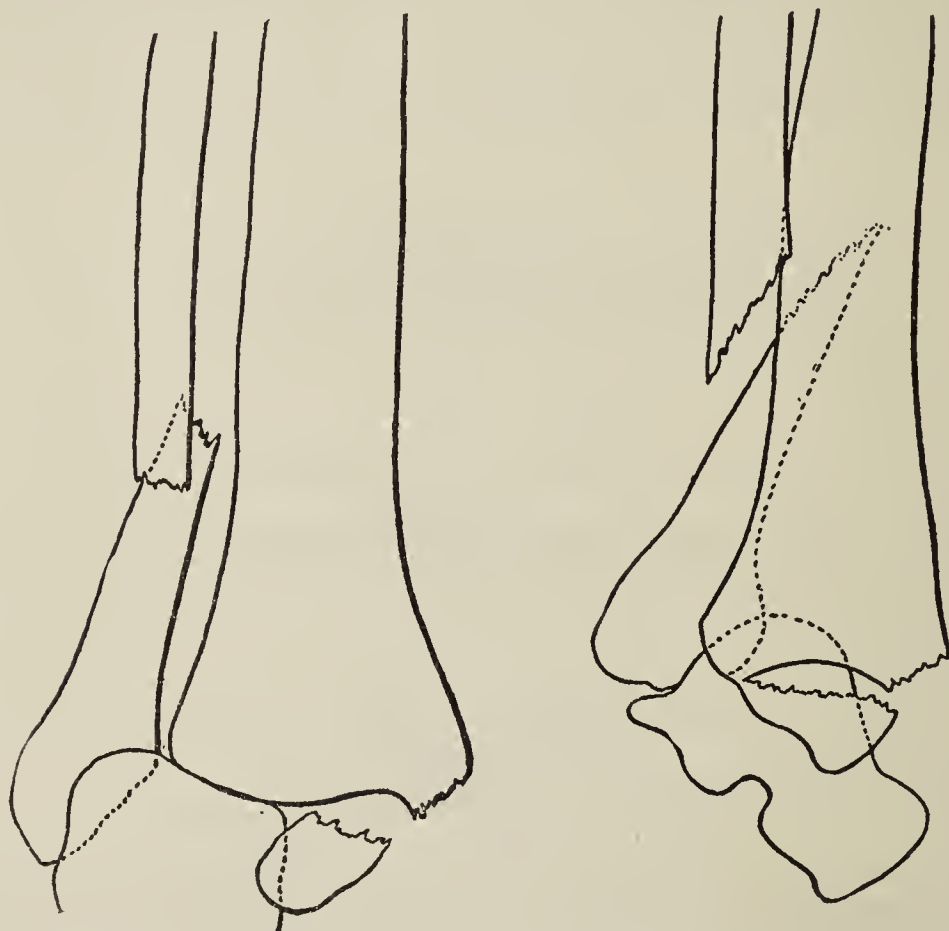


FIG. 25.—Fracture by abduction, second degree (B, II, b) (Pott's fracture, Dupuytren type): a primary fracture of the internal malleolus or rupture of the internal lateral ligament, followed by diastasis (with or without an intermediate fragment), finally succeeded by fracture of the fibula above the inferior tibiofibular joint by bending (Biegungsbruch).

potential moving upper portion, and fracture occurs about 2 to $2\frac{1}{4}$ inches above the malleolus. The line of fracture of the fibula depends to a great extent on whether the fall is directly outward, or whether some torsion of the tibiotarsal joint occurs at the moment of fracture."

On page 590, they describe another (?) mechanism: "In addition to the mechanism described under fibular fracture, there is the effect due to the weight of the body being applied simultaneously with the violence causing the supramalleolar fracture of the fibula; by this force the outer trochlear surface of the astragalus is carried sharply against the outer portion of the articular surface of the

tibia, by external rotation and abduction of the foot, so that either the tibiofibular ligament must give way or the outer edge of the tibia be broken away from the shaft. Dislocation upward of the astragalus between the tibia and fibula may occur."



FIG. 26.—Fracture by abduction, second degree (B, II, b) (Pott's fracture Dupuytren type): diastasis evidenced by detachment of fragment from anterior tubercle of tibia (intermediate fragment); fracture by bending clearly shown by detachment of wedge from concave side of fibula.

Rose and Carless³⁵ (1920) distinguish between Pott's fracture and Dupuytren's fracture thus: "In Pott's fracture, sudden abduction, usually combined with eversion of the foot, results in severe strain on the internal lateral ligament, which gives way, or the base of the internal malleolus is torn off. The astragalus is at the same time driven outward against the external malleolus, and the force is thence transferred up the fibula, which bends and breaks at some weak spot. Generally, eversion is a large element in the force that produces the fracture which then runs obliquely from above downward and forward through the malleolus; less frequently it is due to a pure abduction and may then be situated in the position originally described by Pott, viz., about 3 inches above the tip of the malleolus, and is transverse, the upper end of the lower fragment being displaced inward toward the tibia. The inferior interosseous ligament remains intact."

"In Dupuytren's fracture a much more serious lesion is produced. The interosseous tibiofibular ligament yields more or less completely, or the flake of the tibia to which it is attached is torn off."

Speed³⁶ (1916) thus describes the mechanism of fractures about the ankle (p. 772): "When abduction and eversion of the foot are the cause of the fracture, the astragalus is pushed outward and the fibula tends to break at a point above the termination [presumably he means the *upper* termination] of the tibiofibular ligament in a transverse or oblique line from compressive force. Coincidentally the internal lateral ligament either ruptures or, holding its insertion into the tibia, pulls off the internal malleolus squarely near its lower end (Fig. 566). [This figure represents a "low Dupuytren" fracture.] If this eversion continues strongly, the lower fibular fragment may be separated a little from the tibia by tearing of the tibiofibular ligament, and the internal malleolus is correspondingly dragged outward by the internal lateral ligament and comes to lie under the joint surface (Fig. 567). [This figure shows a Pott's fracture, with wide diastasis; and the latter certainly preceded the fracture of the fibula and did not, as Speed contends, follow it.] Some torsion is present in all these cases. . . . If the torsion is a more predominating feature in conjunction with the eversion, we obtain the spiral fractures of the external malleolus, as this point projects lower down than the internal malleolus and meets with most of the force in external torsion and eversion. [The reason alleged is insufficient explanation.] These spirals, in a quickly-acting force, are above the lower end of the tibiofibular ligament, which by a slight elasticity holds while the rigid bone gives; but in slower-acting force, with more eversion or compression violence from the body weight, the extreme end of the external malleolus is fractured and splintered-up in a spiral manner. As a rule, there is not much damage to the internal malleolus and the internal lateral ligament in this mechanism (see Fig. 568)."

This figure shows a low mixed oblique fracture of the fibula, unreduced, though the foot is in adduction and inward rotation. Speed says reduction has not been secured because of laceration of the external lateral ligament; but how could such a laceration occur in the mechanism he is discussing? Speed adds that "sometimes in eversion, in addition to fibular fracture, the tibiofibular ligament is torn, a condition permitting wide separation between the bone ends and possibly accompanied by a shell of bone pulled out from the tibia." Writing (p. 785) of "malleolar fractures caused by inversion of the foot," he says: "The mechanism of isolated fractures of the internal malleolus is that of fall or compression from body



FIG. 27.—Diastasis of inferior tibiofibular joint, following rupture of internal lateral ligament, but not succeeded by fracture of fibula (B, II, b, 1, variant).

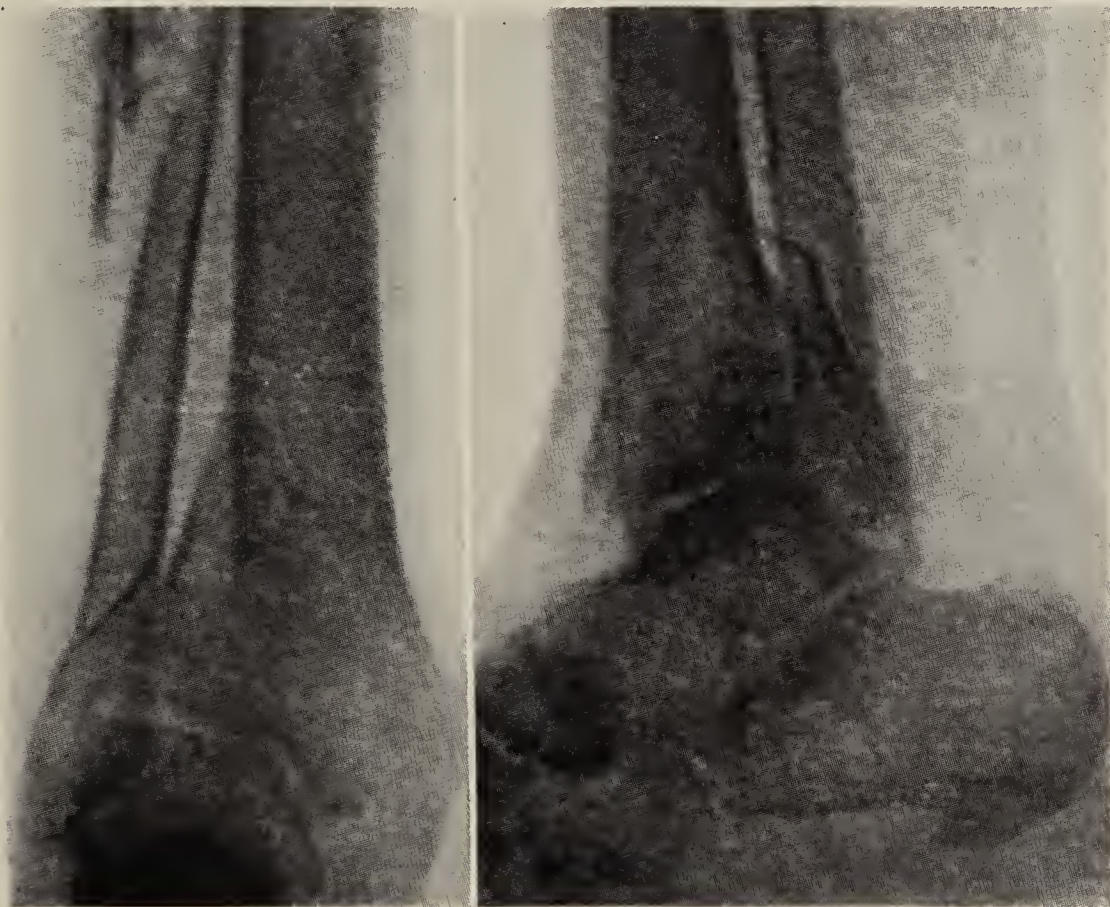


FIG. 29.—Fracture by abduction, third degree (B, III). The internal malleolus is represented by the whole lower end of the tibia; the fibula is broken by bending; there may or may not be an intermediate fragment. There were only two cases of this kind in our series of 300 cases.

weight against the talus, which is tipped inward by the inverted foot. . . . For this result the inversion must not be great, because the pull of the external lateral ligament would also pull off the external malleolus." (Thus he regards fracture of the external malleolus as a secondary and not the primary lesion in adduction fracture. But the illustration he gives as an instance of isolated fracture of the internal malleolus is not happily chosen (Fig. 592), as it shows the astragalus displaced from the external malleolus, an occurrence obviously permitted only by partial rupture of the anterior fibers of the external lateral ligament or sprain fracture of its tip.)



FIG. 28.—Fracture of lower fifth of fibula (surgical neck) by direct violence (kick of horse): there is no diastasis and under such circumstances the typical bending fracture (wedge detached from surface of flexion), which has been overcorrected by adduction of foot, could not have been produced by indirect violence (pressure of astragalus outward on external malleolus).

MECHANISM. Now, after the somewhat tedious historical review given above, it is worth while, before proceeding further in our inquiries, to pause a moment to refresh our knowledge concerning the structure and functions of the ankle. We have read of eversion, of abduction, of rotation, of torsion, compression and bending, of malleolus and fibula, of ligaments and tubercles, of sprains, displacements and diastases; but it is clear that few authors use any of these terms in the same

senses, and that some of them probably do not know what they mean themselves. But what Nélaton³⁷ (1844) said of surgery in general may well be applied to this particular part of surgical knowledge: "*Rien en chirurgie n' est assez abstrait pour que*

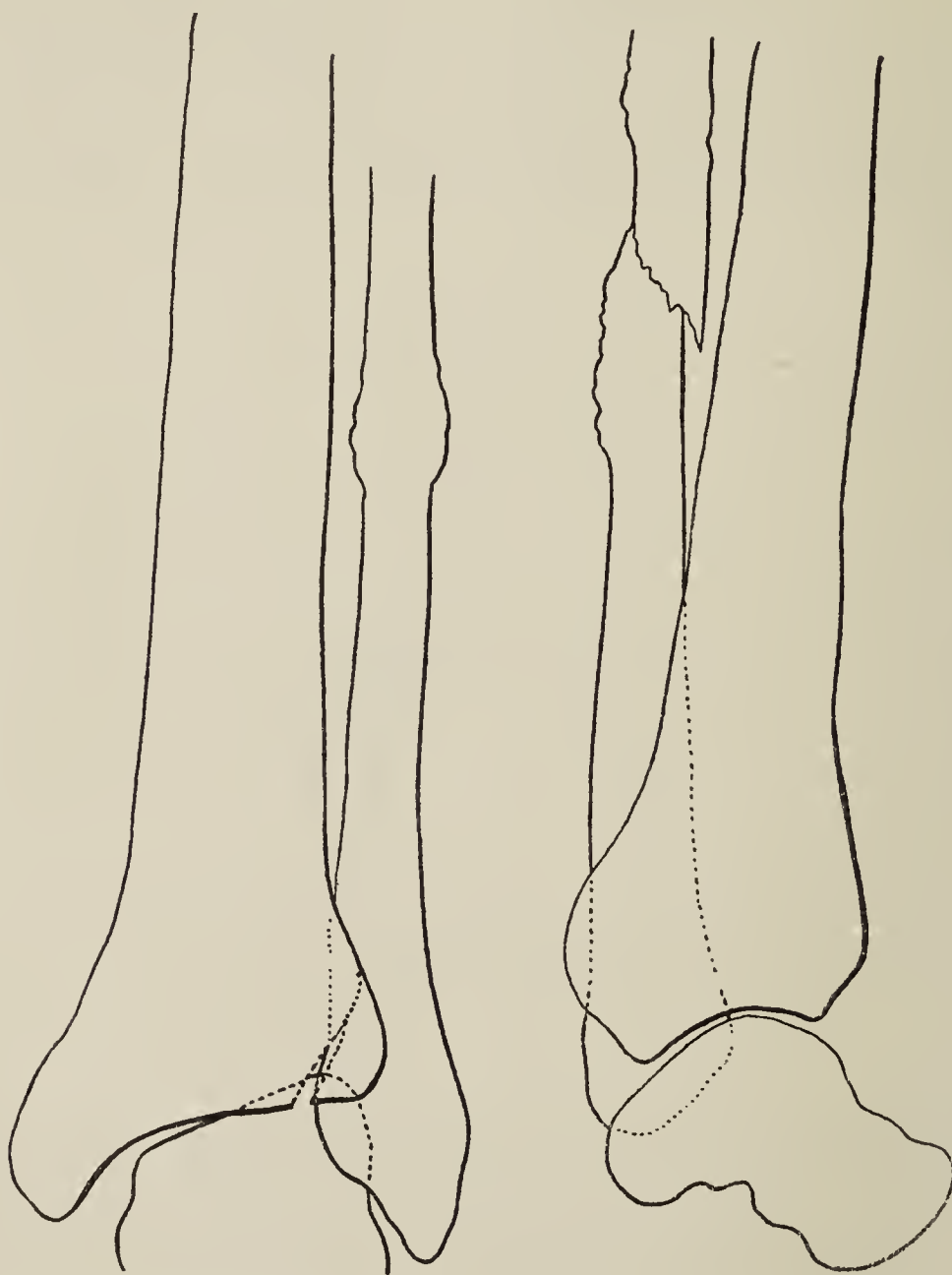


FIG. 30.—Fracture by abduction (B, II, b, 1): rupture of internal lateral ligament, followed by diastasis (note the intermediate fragment), and this succeeded by a bending fracture of fibula, in this case at an unusually high level.

l'obscurité s'excuse par le sujet même, et sur un grand nombre de points la science est assez avancée pour que la vérité apparaisse au milieu des controverses à celui qui la cherche sans préoccupation." And if what I succeed in explaining in the following pages appears to you to be nothing new, you will admit its truth;

and I shall be satisfied. If on the other hand, you do not agree with my conclusions, that will not in the least disconcert me, for it will not impair their truth.



FIG. 31.—Fracture of posterior margin of tibia, associated with a tibiofibular diastasis, as evidenced by sprain fracture at this joint. The posterior marginal fragment which is unusually large is visible also in the anteroposterior view.

The ankle-joint is formed above by the tibiofibular mortise, and below by the trochlea of the astragalus, which fits into the mortise as a tenon. This trochlear surface is one-fourth wider in front than behind, conforming to the divergent direction of the internal surfaces of the malleoli. The inferior articular surface of the tibia may be described as the roof or ceiling (*plafond*) of the joint; and the articular surfaces of the malleoli have long been known as the “cheeks” of the mortise.³⁸ That portion of the fibula *which projects beyond the tibial plafond* is properly called the external malleolus, and the corresponding projection of the tibia is called the internal malleolus. The posterior lip of the tibial plafond projects so low as to have been called by Destot the posterior malleolus. This serves

to reinforce the mortise posteriorly. It is further deepened by the transverse tibiofibular ligament which extends from the external malleolus to the posterior lip of the tibia. Thus in walking, as the foot meets the ground in plantar flexion, the leg bones are checked in their tendency to slide forward on the astragalus by the wedge shape of the trochlea of the latter bone (broad anteriorly and narrow posteriorly), by the corresponding divergence of the anteroposterior planes of the malleoli and by the long posterior lip of the tibial plafond. In walking backward (always digitigrade, not plantigrade) the same mechanism is effective.

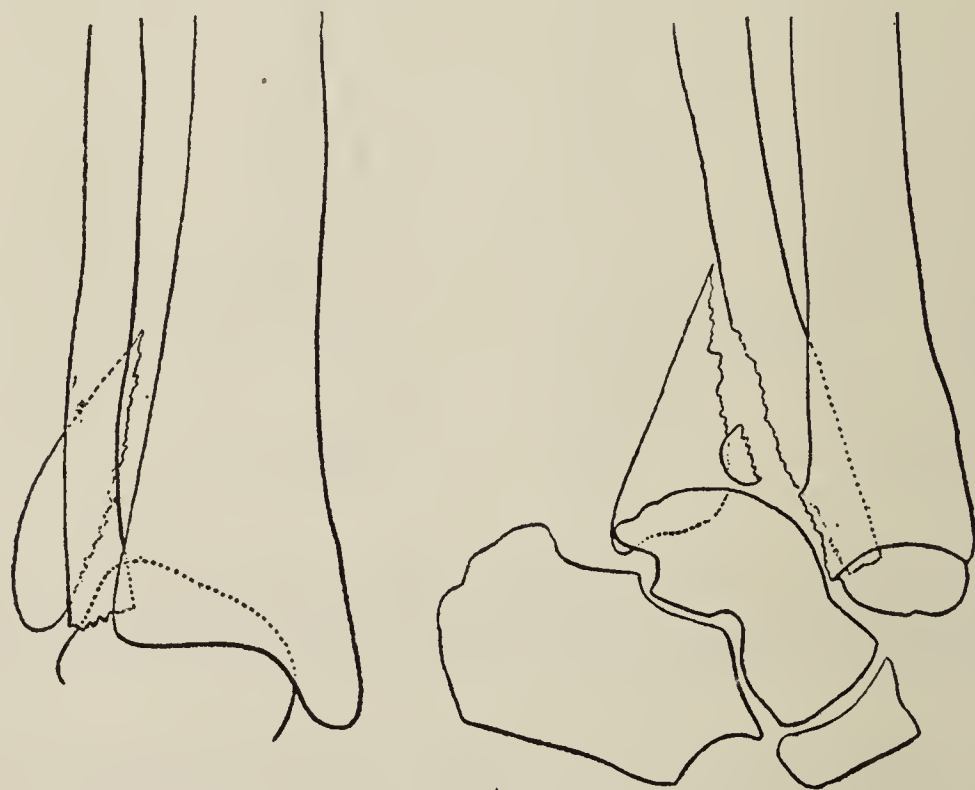


FIG. 32.—Small posterior marginal fragment in a case of fracture by external rotation. Notice the mixed oblique fracture of fibula; the intact internal malleolus (rupture of internal lateral ligament) and the complete posterior dislocation of the foot, the astragalus and the posterior marginal fragment accompanying the external malleolus.

The fibula is attached firmly to the tibia, but a slight range of motion is permitted. The interosseous membrane extends throughout the length of the tibial and fibular shafts, the fibers running downward and laterally from tibia to fibula (as in the forearm from radius to ulna); and, in addition, there are strong ligaments whose fibers run in the same direction, binding both ends of the fibula to the tibia, at which points only are the two

bones in contact. At the upper end, where the head of the fibula butts against the overhanging external condyle of the tibia, there is a synovial cavity to the tibiofibular joint; but at the lower end, where the fibula is received into a longitudinal groove between the anterior and posterior tubercles on the lateral surface of the tibia (Fig. 4), no such joint cavity exists, union being effected by a dense feltlike interosseous ligament, reinforced anteriorly by the anterior inferior tibiofibular ligament, and posteriorly by the posterior ligament of the same

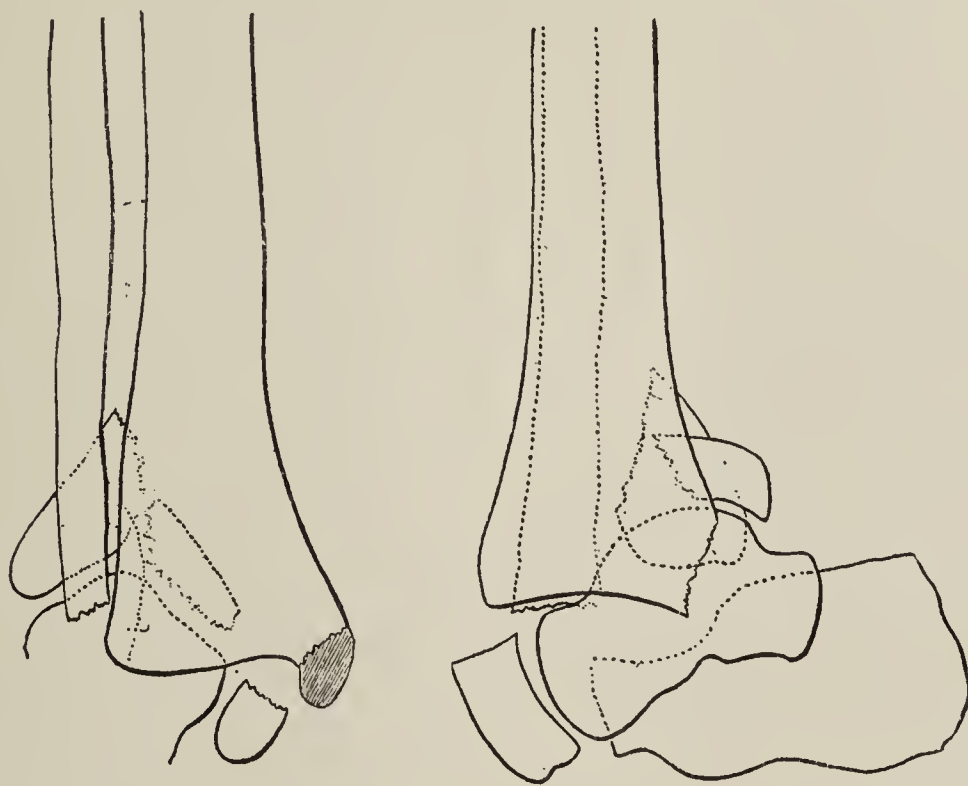


FIG. 33.—Medium-sized posterior marginal fragment in association with mixed oblique fracture of fibula and fracture of internal malleolus. The posterior marginal fragment, which is visible also in the anteroposterior view, accompanies the external malleolus and the astragalus in the very marked lateral displacement and in the incomplete posterior dislocation.

name. The malleoli, of which the external is the longer and is situated more posteriorly, serve to keep the foot (which is appended to the astragalus) under the leg bones. The astragalus itself has no muscles attached to it, and serves only as a ball in a ballbearing joint to facilitate movements of the leg bones above it and of the tarsal bones below and in front. The foot is attached to the leg bones by ligaments, of which the lateral portions are best developed, constituting for the ankle, as in other hinge joints, lateral ligaments which hinder motion

except in the anteroposterior plane. The internal lateral ligament passes from the internal malleolus in radiating direction (1) anteriorly to the scaphoid and median surface of the calcaneum (*sustentaculum tali*) and (2) posteriorly to the median tubercle on the posterior surface of the astragalus. The external lateral ligament has three distinct bands, passing from the external malleolus: one goes forward to the lateral border of the neck of the astragalus, just above the sinus of the

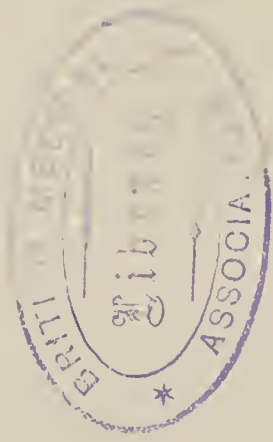


FIG. 35.—Small posterior marginal fragment in association with abduction fracture of fibula and fracture of internal malleolus. Note the diastasis, the typical wedge detached from the flexion surface of fibula and the posterior marginal fragment accompanying the external malleolus and astragalus in their marked lateral and posterior displacement.

tarsus; the middle band passes downward and slightly backward to the calcaneum; while the posterior, whose deep portion is extremely strong (Fig. 5), is attached to the lateral tubercle on the posterior surface of the astragalus (*os trigonum*), which, being from 5 to 7 mm. posterior to the median tubercle, is the portion of the astragalus which casts the farthest posterior shadow in lateral roentgenograms of the foot. This posterior band of the external lateral ligament is so exceedingly strong that it is very seldom ruptured; it holds the astragalus almost



FIG. 34.—Large posterior marginal fragment in association with mixed oblique fracture of fibula and fracture of internal malleolus; only moderate displacement, chiefly due to the outward rotation.



indissolubly attached to the external malleolus,³⁹ and in injuries of the ankle either one or other bone to which the ligament is attached is more easily broken than is the ligament ruptured. As will be shown subsequently, usually the fibula gives way when the strain comes; occasionally, however, the posterior tubercle of the astragalus is detached; and I have seen at least one case in which fracture occurred at both points simultaneously.⁴⁰



FIG. 36.—Posterior marginal fragment associated with diastasis and fracture of internal malleolus, and with fracture of fibula by bending backward (flexion surface posterior, extension surface anterior) as evidenced by wedge detached from posterior surface of fibula.

The next question that arises is, what is the function of the fibula?

Humphry⁴¹ (1858) notes that the fibula is an inconstant bone in animals: in carnivora and pachydermata it extends from the upper end of the tibia to the ankle, as in man. In most rodents it is united with the tibia at the lower part. In ruminants it alto-

gether disappears. In birds its upper extremity enters into the knee-joint and articulates with the external condyle of the femur; it lies close against the tibia and dwindles and disappears about the middle of the leg. In reptiles, it is large, in many extending to the knee-joint above and to the tarsus below. In the bat, the lower half of the fibula and the upper half of the ulna are retained.

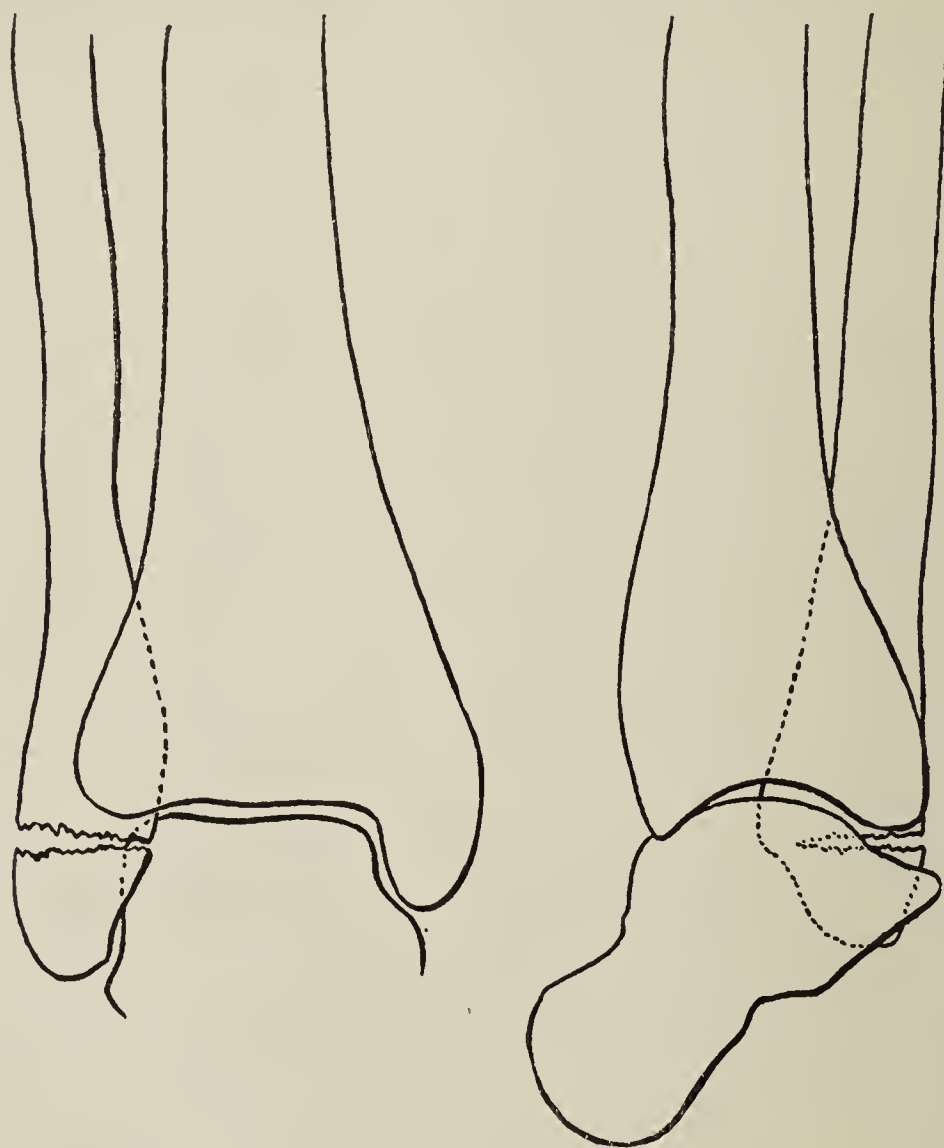


FIG. 37.—Fracture by adduction, first degree (C, I); avulsion of external malleolus. (See page 322.)

It has seemed to me, from study of the skeletons in the Museum of the Academy of Natural Sciences of Philadelphia, that the fibula was best developed and extended farthest beyond the tibia at the ankle in those animals which were most nearly plantigrade, and in which, as in man, stability rather than agility was demanded. In digitigrades, such as the horse and camel, there is no fibula; in partial or less complete digitigrades (rhinoceros) it extends beyond the tibial plafond, but not below the level of the internal malleolus. In a still less complete digitigrade (almost a plantigrade), such as

the elephant, it extends below the level of the internal malleolus, and there is outward rotation of the lower end of the tibia, as in man. In the gorilla, chimpanzee, orang-utan, etc., on the other hand, which are more plantigrade than digitigrade (but in which, as already noted, agility is retained at the expense of stability) the lower end of the tibia has not rotated out as far as in man (in fact, not so far out as a transverse plane through the tibial condyles), and the fibula descends no lower than the internal malleolus. This lack of development of the fibula in these more or less anthropoid

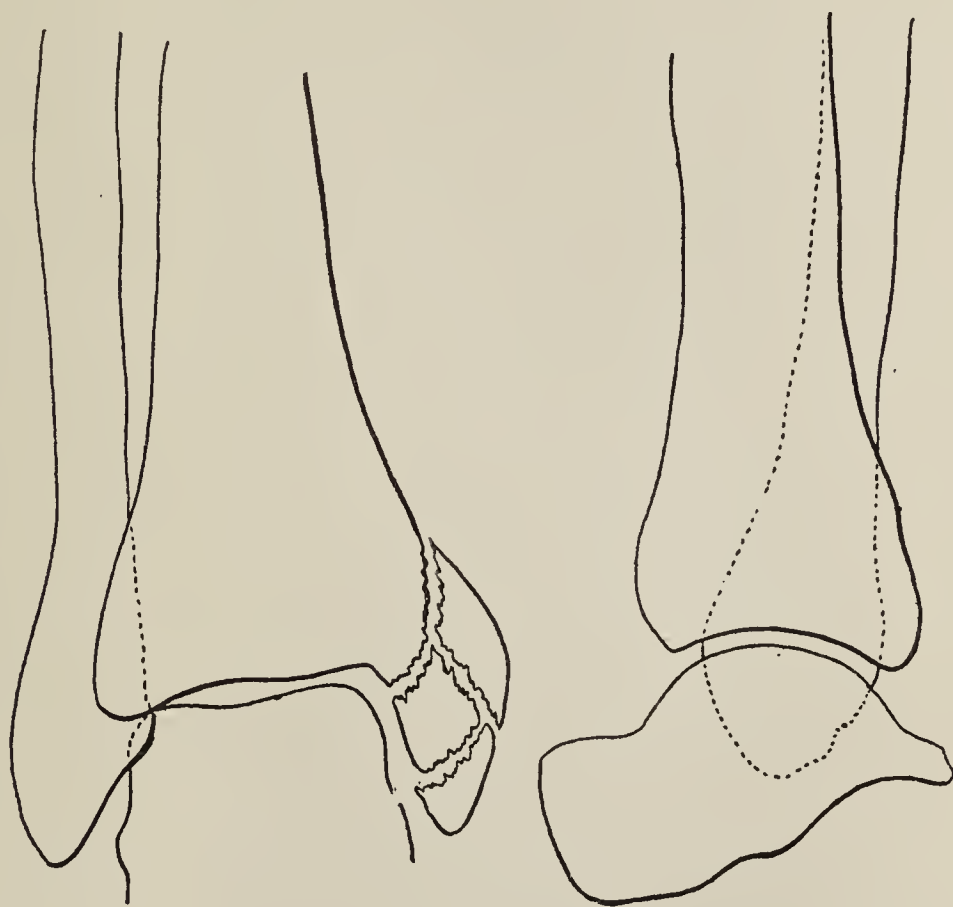


FIG. 38.—Adduction fracture, second degree (C, II, a); avulsion of external malleolus, represented by partial rupture of anterior fibers of external lateral ligament (rare) followed by compression fracture (note the comminution) of internal malleolus.

animals was noted by Bland-Sutton⁴² (1888); and he further calls attention to the fact that in babies born with congenital clubfeet this deformity is merely a lack of normal development, the outward rotation of the lower end of the tibia not having occurred, and the external malleolus not having descended below the level of the internal. Evidently, he concluded, the external malleolus was developed only to aid members of the human race to walk in the erect posture; it was required to keep the foot steady and prevent it from turning outward into a position of extreme valgus. As

Destot says, the external malleolus acts merely as a splint to maintain the direction of the foot.

But if this is so, why is the fibula a separate bone in man? Why is not the external malleolus merely a part of the tibia? To this I know of no better answer than that given by Bromfeild¹¹ (1773), that if it were a part of the tibia, and no give or play whatever occurred between the malleoli, no one could take more than a very few steps without fracturing one or other malleolus.

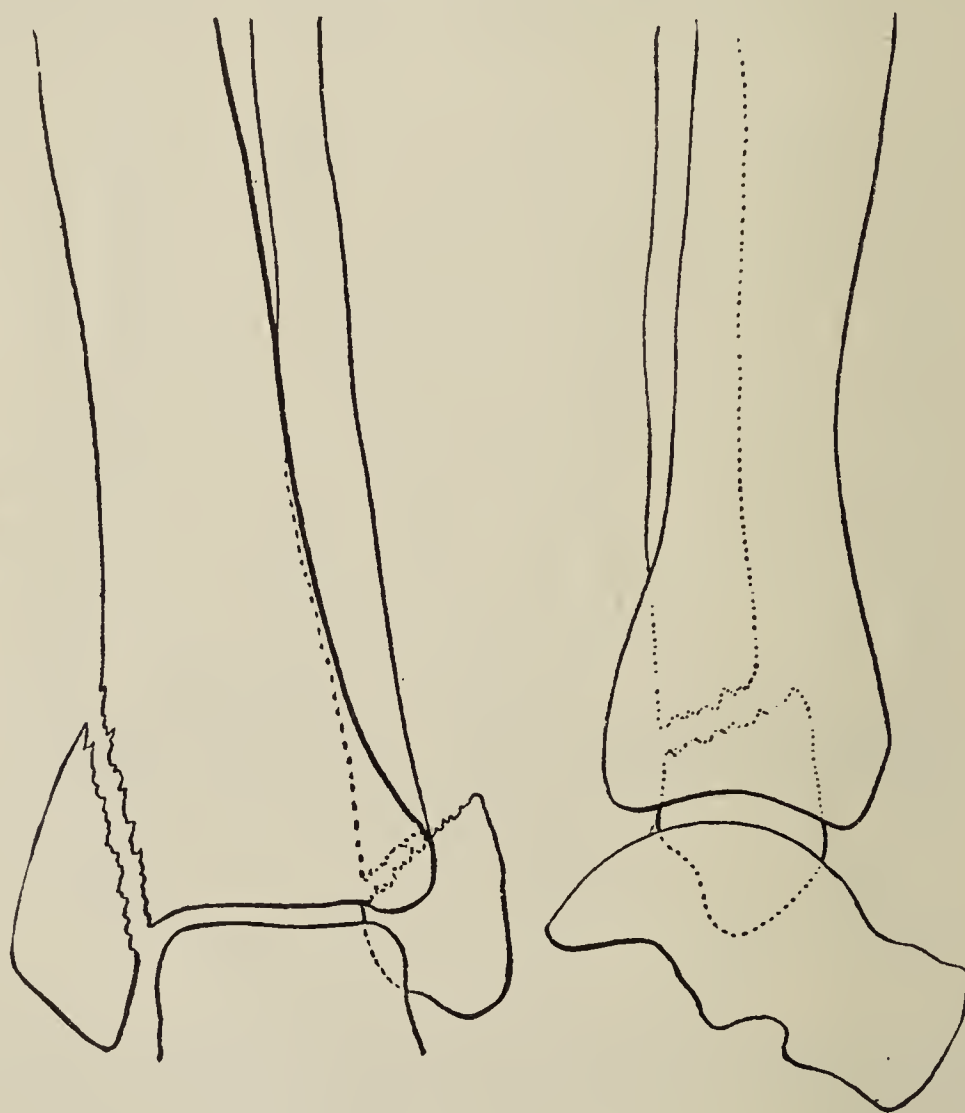


FIG. 39.—Adduction fracture, second degree (C, II, a); avulsion of external malleolus followed by compression fracture of internal malleolus.

If a leg is studied from which all soft parts have been removed except the ligaments and the interosseous membrane, these phenomena may be observed:

In full plantar flexion (extension) of the foot, the anterior and middle bands of the external lateral ligament become tense and pull the external malleolus medially (and slightly downward and backward) against the tibia, keeping the external malleolus in close contact with the astragalus as this glides forward and presents to



FIG. 40.—Adduction fracture, second degree (C, II, b); large tibial fragment extending into shaft replaces compression fracture of internal malleolus.

the intermalleolar space a slightly less diameter than in full dorsiflexion of the foot. In the latter movement, dorsiflexion, the anterior band of the external lateral ligament becomes lax, and as the external malleolus is forced away from the tibia and slightly upward, the anterior and posterior tibiofibular ligaments become tense, especially the anterior ligament; also, as flexion beyond a right angle occurs, much tension develops on the middle band of



FIG. 41.—Adduction fracture, second degree (C, II, b); splitting fracture of median articular surface of tibia replaces crush of internal malleolus. (See page 322.)

the external lateral ligament, and this pulls the malleolus backward: the posterior band of the external lateral ligament is always tense; it makes the astragalus and external malleolus practically one bone.

The expansion of the intermalleolar space which occurs during dorsiflexion of the foot may amount to several millimeters. I have measured it by affixing a wire in each malleolus and bending the

ends of these wires forward over the ankle-joint until they crossed each other in parallel lines: by scratching a mark on each wire at the same point when the foot is in full plantar flexion, it is easy to measure the excursion as the foot is brought up into full dorsiflexion. This excursion, which exceeds 2 mm. and may approach 3 mm., allows the wider anterior diameter of the articular surface of the astragalus to pass back between the malleoli in dorsiflexion, while the downward drag of the anterior and middle bands of the



FIG. 42.—Adduction fracture, third degree (C, III); detachment of lower epiphysis with splitting of median surface of tibia.

external lateral ligament keeps the malleoli in contact with the small posterior diameter of the astragalus, which presents between them in full plantar flexion. Destot⁸ (1911) pointed out that, inasmuch as the intermalleolar axis and the axis of rotation of the astragalus in flexion and extension do not coincide (they form an angle of about 30 degrees, open laterally, Fig. 4), it is not a directly transverse diameter of the astragalus that presents between the malleoli at any point of flexion or extension, but a diameter of

varying obliquity, which is, however, always nearly the same in length; and he is inclined to ignore the existence of the movements of the external malleolus. And I have dwelt at some length on these movements because I have found them nowhere described and because there has been some dispute about them. Humphry⁴¹ (*loc. cit.*, p. 557) asserted that the increase of the distance between the malleoli was secured solely by the elasticity of the fibula, which



FIG. 43.—Adduction fracture, third degree (C, III); the supramalleolar fracture by adduction (type produced experimentally by Tillaux). (See pages 279 and 323.)

bent inward toward the tibia in its lower fourth, when the external malleolus was forced outward. Nancrede⁴³ (1880), however, pointed out that an upward and downward movement of the fibula occurred, and asserted that Humphry's theory was "preposterous and untrue."⁴⁴ This is my own opinion, also.

The upper end of the fibula can also be seen to move in flexion and extension of the foot at the ankle; in full plantar flexion (with medium and downward movement of the external malleolus), the

superior end of the fibula moves slightly forward and rotates slightly outward, its anterior surface turning away from the tibia. This movement is due largely to the median and backward pull exerted on the external malleolus by the middle and posterior bands of the external lateral ligament. The head of the fibula slides backward and very slightly upward again in full dorsiflexion. Thus the chief movement of the superior tibiofibular joint is an anteroposterior one (downward and forward, or upward and backward) around the inferior tibiofibular joint as a pivot; but these movements are so slight as to be scarcely appreciable.

Very little change occurs in the interosseous membrane during these movements, except in its lower fourth, where it spreads and becomes tense (the aperture for the anterior peroneal artery tends to become round from oval) as the external malleolus ascends and moves backward; and it again becomes relaxed when the external malleolus descends and moves mesially toward the tibia. The strength of the interosseous membrane is much greater than usually supposed. Even when the lower end of the fibula is freed from its tibial attachments, very great force is required to rupture the interosseous membrane, and fracture of the fibula in its lower third is the nearly invariable sequel.

The normal movements of the ankle-joint are those of flexion and extension—20 degrees of dorsiflexion and 60 degrees extension or plantar flexion, a total of 80 degrees, approximately. This motion occurs around an axis which passes in the frontal plane somewhat below and in front of the tip of the external malleolus. This axis makes an angle of 30 degrees (thereabouts) with the bimalleolar axis. This arrangement accounts for the greater excursion forward of the lateral astragalar surface in relation to the external malleolus as compared with the motion which occurs between the median surface of the astragalus and the internal malleolus; as well as for the apparent deviation of the point of the foot medially in full plantar flexion, and laterally in full dorsiflexion.

If movements were possible in the ankle-joint around an anteroposterior axis, they should be named adduction (tibial flexion) and abduction (fibular flexion), or movements in the frontal plane toward and away from the median line. These movements are quickly resisted by the tension on the lateral ligaments (Figs. 6 and 7), and if forced, the malleolus *away*

from which motion occurs is torn off by its ligament, or the ligament itself ruptures (see the experiments of Hönigschmied, related at page 279). Motions of adduction and abduction in the foot normally occur in the subastragalar joint and permit the calcaneum without difficulty to accommodate itself to slight irregularities of the soil. But the calcaneum is attached to the astragalus by the extremely strong astragalocalcanean interosseous ligament; and when such movements are too extensive, they are transmitted directly to the astragalus and from it to the tibiofibular mortise where fracture of one or the other malleolus is the usual consequence. Not until that malleolus on which the pull comes has given way, or the corresponding ligament has ruptured, is the astragalus able to act on the other malleolus by a push so as to produce a compression fracture. Rare exceptions to this general rule occur, however, as when the calcaneum is itself first broken, and becomes so displaced as to press directly on the end of the fibula, producing a compression fracture of the external malleolus (Fig. 23).

Movements of rotation around the long axis of the leg may be attempted in the ankle-joint by twisting the point of the foot toward or away from the median line. As pointed out by Maisonneuve⁴ (1840), movements of inward rotation are almost inseparable from a movement of adduction, as the numerous joints in the anterior tarsus render the foot very mobile in this direction. Any movement toward outward rotation, however, converts the foot into a rigid lever, and motion is easily and with much force transmitted to the ankle-joint, the astragalus attempting to turn so as to bring its long axis crosswise between the malleoli. Owing to physical laws, it is on the external malleolus that the greatest strain comes. Maisonneuve illustrated this by placing a ruler (which represents the foot) between two parallel volumes (which represent the malleoli): the volume which is moved is always that toward which the long end of the lever moves (Fig. 8); even if this volume be much heavier than the other it is easily moved by the greater leverage exerted. In movements of outward rotation, the foot, relatively to the tibia, is a lever of the first order, with its fulcrum on the anterior border of the

fibula: the arm of the resistance will have, say, a length of 3 cm., that of the power 12 cm. (the length of the foot being taken as 15 cm. from the toes to the posterior border of the ankle-joint). Relatively to the fibula, it is a lever of the second order, with its fulcrum at the posterior border of the internal malleolus: the resistance, therefore, has an arm of 3 cm. (as in the other case) but the power has an arm of 15 cm. (the whole length from the point of the foot to the fulcrum). Thus the force which tends to fracture the fibula is as 12 is to 9, or as

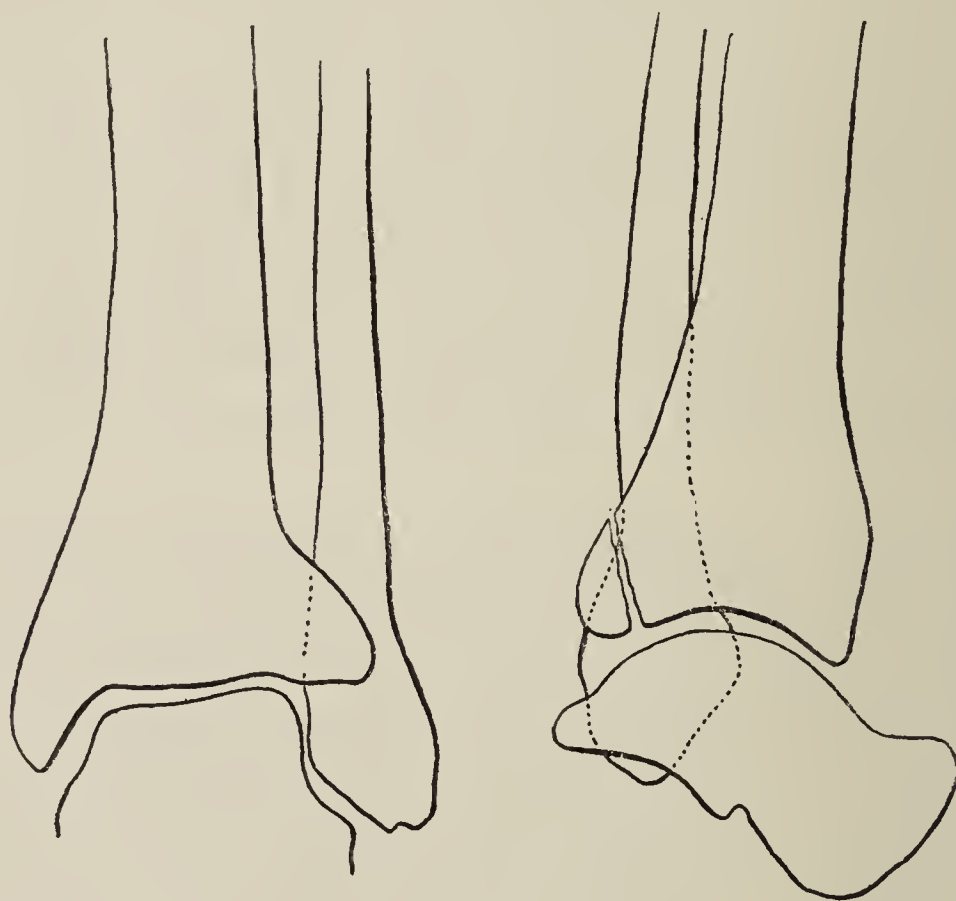


FIG. 44.—Isolated fracture of posterior tibial margin; no displacement; from compression upward and backward.

4 is to 3. Such a mechanism as this (outward rotation of the foot) usually causes an oblique fracture of the lower end of the fibula (see Hönigschmied's⁶ experiments, page 279); and if such a fracture be made by an osteotome it will be found that *external rotation is the only movement that will cause separation of the fragments* (Fig. 9). It is evident that the mechanism of this fracture, which is the most frequent of all fractures of the ankle (more than 25 per cent of all cases), involves not only a push outward on the anterior border of the external malleolus,

as noted by Maisonneuve, but also, as Hönigschmied pointed out, a pull inward on its posterior border by means of the posterior band of the external lateral ligament. The line of this fracture is oblique from above and behind, downward and forward. It is, properly speaking, a spiral fracture produced by torsion. Its obliquity varies greatly; but it is always higher on the posterior surface of the fibula than on its anterior, and the line of fracture passes through and involves the inferior tibiofibular joint. Almost invariably, its lower and anterior end extends to the external malleolus (in 90 per cent of our cases): often just below the tibial plafond, sometimes as far down as the very tip of the malleolus. Thus in practically every instance the anterior inferior tibiofibular ligament remains intact, or even if partially ruptured, there results no true diastasis between fibula and tibia. At most, the lower fragment, comprising that part of the fibula posterior to the attachment of the anterior tibiofibular ligament, rolls outward and slightly backward around the unruptured posterior tibiofibular ligament as a hinge.

If this "mixed oblique"⁴⁵ fracture of the fibula, as Destot names it, is the sole lesion resulting from outward rotation of the foot, there is little or no displacement (Figs. 10 and 11). This was the case in all of the 79 cases studied by Dr. Bromer and myself. If the force continues to act, the next lesion which is added is rupture of the internal lateral ligament (in 12 cases only, in our series), or, far more frequently, fracture of the internal malleolus, usually only of its anterior tip, seldom of its whole extent (Fig. 12). This combined lesion (oblique fracture of the fibula with fracture of the internal malleolus) occurred in 32 cases in our series, or in 10 per cent of the entire number.⁴⁶ The displacement may be slight or marked. And in very many cases (51 additional cases in our series), besides these two lesions, there is added the complication of fracture of the posterior margin of the tibia. Counting in all complications and variations, this type of fracture occurred in 100 cases or in 33 per cent of the total 300 cases we have studied.

Seldom does the obliquity of the fibular fracture pass so high as to be above the level of the anterior inferior tubercle of the tibia (Figs. 13 and 14): in 3 cases this tubercle was

detached (in 2 of these there was no appreciable lesion at the internal malleolus, but in 1, the internal malleolus was fractured), and in 2 others it is probable that a disjunction of the joint had occurred, as indicated by suggestive roentgen-ray findings. This lesion (fracture of the anterior tibial tubercle, or diastasis of the tibiofibular joint) must occur previous to, even if nearly simultaneously with, the oblique fracture of the

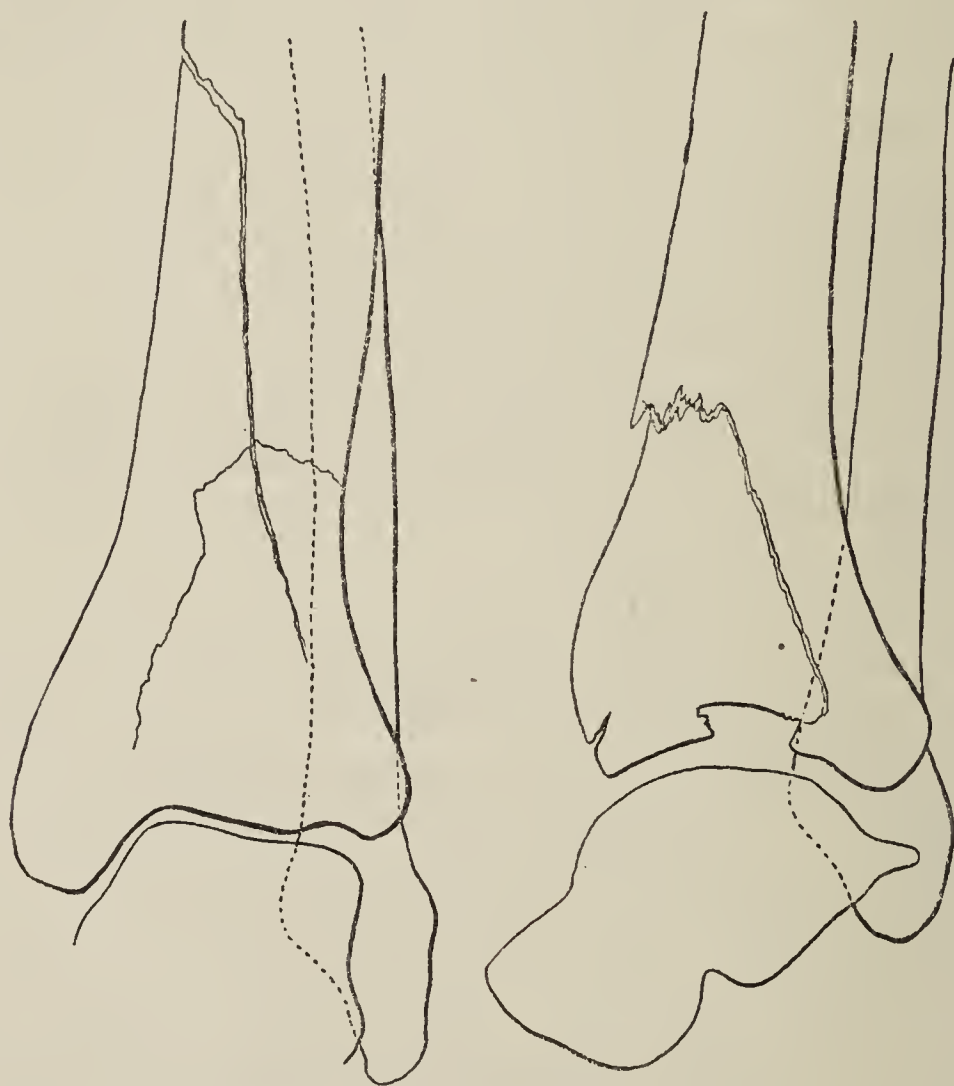


FIG. 45.—Comminuted fracture of tibial plafond; from compression upward.

fibula;⁴⁷ because after the fibula is fractured its lower fragment (on which alone the force is acting) is already detached from the anterior tubercle of the tibia, and a continuance of the force would merely increase the separation. Moreover, there is lack of displacement in these isolated oblique fractures of the fibula merely because to permit displacement it is necessary that the internal malleolus (or its ligament) previously give way. If a tibiofibular diastasis (with or without separa-

tion of the tubercle) occurs (and if it occurs it must always occur previous to a fracture of the fibula, as already remarked), then the fracture of the fibula (by torsion still) occurs not through the inferior tibiofibular joint but above it, sometimes through its surgical neck, often through its true neck in the upper third of the fibula, as originally pointed out by Maisonneuve. That such a lesion in the upper third of the fibula can occur without appreciable bony lesion at the ankle cannot be denied. We have two such cases; and Quénu⁴⁸ went so far as to say that many more such fractures occur without diastasis than with it; and that they may occur even without any lesion at all at the ankle-joint (Fig. 15).⁴⁹ It must also be recognized that diastasis is not necessarily followed by fracture of the fibula at any level, as shown in the lesion represented in Figs. 16 and 17 in which the fibula detached the anterior inferior tubercle of the tibia, and in which, after rupture of the internal lateral ligament had occurred, the fibula was forced by the astragalus around back of the tibia by continuance of external rotation of the foot, as in the mechanism described by Huguier⁵⁰ (1848), though in his cadaveric experiments, as well as in the case illustrated by Destot (Fig. 67, p. 142 of his monograph), this displacement was accompanied by (and I believe succeeded by) a fracture of the upper end of the fibula.

In rare instances, the avulsing force on the internal malleolus may be so great as to cause fracture of the entire lower end of the tibia (or in children a separation of the epiphysis⁵¹): this appears to have been the mechanism in four of our cases, which on a purely anatomic classification should perhaps be grouped with the supramalleolar fractures (Figs. 18 and 19).

The significance of the posterior marginal fragment, and the mechanism by which it probably is produced, will be discussed in another place (p. 317).

I desire to return now to the movement of *forced abduction*, a discussion of which has been intentionally postponed until disposition had been made of the much more frequent mechanism (outward rotation). We find in any large series of fractures at the ankle a certain number in which fracture of the internal malleolus is the only lesion (in our series this lesion

occurred in more than 6.5 per cent of the whole number). Now it is not rational to suppose that the same mechanism which at one time causes an isolated oblique fracture of the lower end of the fibula will at another cause an isolated fracture of the internal malleolus: they must be produced by different mechanisms. Experimentally, it is very clearly seen (note the experiments of Hönigschmied, detailed on page 279, that straight abduction (fibular flexion) of the foot has as its primary and most constant lesion fracture of the internal malleolus, or its equivalent, rupture of the internal lateral ligament. This is

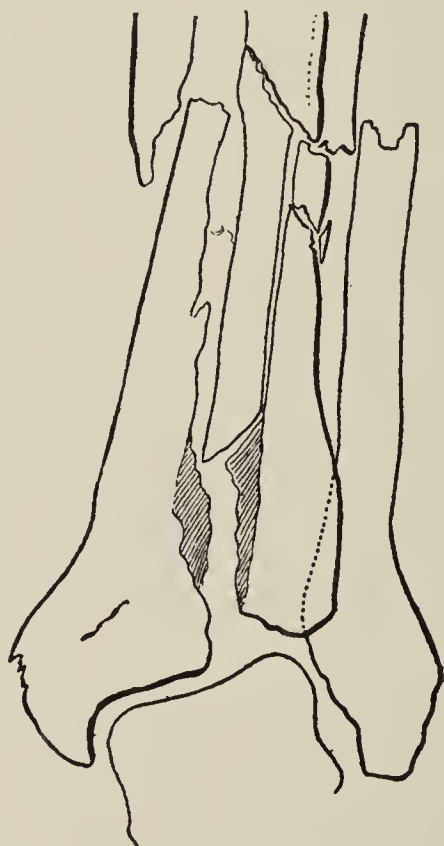


FIG. 46.—T or Y fracture involving ankle-joint; from compression upward.

a prerequisite in order to free the astragalus sufficiently so that it may press directly on the external malleolus, which it does by rotation around an anteroposterior axis. The experiments of Bonnet⁵² (1845), have been overlooked by most students. He showed, long before Tillaux or Hönigschmied, that abduction (fibular flexion) of the leg, while the foot was held in a vise, caused: first, fracture of the internal malleolus or rupture of the internal lateral ligament (Fig. 20), and, if the abduction was increased, a crushing or fracture of the external malleolus (Fig. 21), never of the fibula above the

inferior tibiofibular joint. But sometimes no lesion of the external malleolus was caused even when the internal malleolus was widely separated. If, on the other hand, abduction of the foot was produced with the leg lying on its fibular side, but with the foot projecting free of the table, the same lesions occurred at the internal malleolus; but the fibula broke above the inferior tibiofibular ligaments at the point where it rested on the table.

In the mechanism of these fractures by abduction the influence of the tibiofibular ligaments is paramount:

1. If the tibiofibular ligaments hold, the fibula breaks across through the external malleolus proper (*i. e.*, below the tibiofibular ligaments) and not above these ligaments by that "preposterous and untrue" mechanism to which Nancrede objected, namely, the inward bending of the fibula toward the tibia. It is not proper, perhaps, to deny that the latter mechanism might sometimes occur (all things are possible) in a patient with exceedingly relaxed ligaments; but I feel strongly inclined to state in the words of Souligoux (applied by him to the existence of an isolated fracture of the posterior tibial margin) that I do not believe such a mechanism exists, and will not believe it exists until somebody shows me its method of production. Of the 300 fractures which we have studied, we find only 13 cases which seem to belong to this type (bimalleolar fracture by abduction): evidently the more nearly the movement of the foot conforms to the type of straight abduction, the more apt is diastasis, and as a consequence, fracture above the tibiofibular ligaments to occur (see below); while the more nearly it corresponds to external rotation (deviation of the point of the foot outward), the more certain is the fracture to be oblique in type, involving the inferior tibiofibular joint, but, as already explained, causing no true diastasis; hence the extreme rarity of true bimalleolar fractures by abduction (Fig. 22).⁵³

2. If the tibiofibular ligaments rupture, then the fibula is freed from the tibia, and if the force continues (in life it is now the weight of the body borne chiefly, or at least abnormally, on the fibula), the fibula breaks "by flexion" and the break usually occurs where the fibula is weakest, through the

surgical neck above the inferior tibiofibular ligaments. Study of roentgenograms or museum specimens of fractures of this type shows clearly two things: an evident tibiofibular diastasis, and a flexion fracture (*Biegungsbruch*) of the fibula. The typical mechanism of the flexion fracture is illustrated in Fig. 24 (p. 287), copied from Messerer; and though at first glance some roentgenograms of fractures of the fibula at this height may not seem to indicate this mechanism clearly, more careful study (in nearly every case, at least, with which I am familiar) shows that the fracture conforms to the *Biegungsbruch* type:



FIG. 47.—T or Y fracture involving ankle-joint; from compression upward.

thus the line of fracture is either nearly transverse (rare), slightly oblique (frequent, Fig. 25), or (very frequent) is comminuted in the typical manner, with detachment of a wedged shaped fragment from the concavity of the bent bone (Figs. 26 and 35); that is to say, the wedge is on the lateral border of the fibula (apex toward the tibia) if the fibula was broken by straight abduction of its lower end, or on the posterior border (apex anteriorly) if it broke by posterior displacement of its lower end (Fig. 36).

Now it is a prerequisite for a bone (or any other similarly

shaped structure) to be broken by indirect force through bending that one of its ends must be fixed and the other end movable. The upper end of the fibula is fixed by its attachments at the superior tibiofibular joint and by the interosseous membrane; and to permit fracture by bending of its shaft, under such circumstances, by means of indirect force applied to the external malleolus, it is first of all necessary that the lower end of the fibula be freed from its attachments to the tibia. If these attachments are not freed, it is extremely unlikely that a fracture by bending can occur; though it is possible to conceive of an exceptional instance in which such an event might occur, as, for instance, if the surgical neck of the fibula (as in the second mechanism described by Bonnet) were pressed against the edge of a table or similar object by abduction of the foot; but in such a case the action of direct violence in causing the fracture could not be excluded. In life it would be less unlikely for a fracture by compression to occur (the force being transmitted in the long axis of the fibula which was still rigidly attached to the unbroken tibial shaft); whereupon the fracture would present a very different appearance (Fig. 15); or even less unlikely for a fracture by torsion to occur, though for this mechanism also it is necessary for the two ends of a bone to be movable in opposite directions or for one to be fixed and the other movable. Torsion was held by Maisonneuve (and no doubt correctly) to be the mechanism by which was produced the fracture in the upper third of the fibula described by him as "fracture par diastasis;" and it is this mechanism (torsion) which causes it to be situated so close to the fixed end of the bone,⁵⁴ and which makes me class it as a variant of the fracture "by outward rotation of the foot" already discussed; and which convinces me that it does not belong in the same class with fractures produced by straight abduction of the foot, in which cases, I repeat, the fracture of the fibula occurs in its lower third and is caused by a bending mechanism.

It is true, of course, that the force may cease to act before the fibula breaks; in which case merely a diastasis results (Fig. 27).

Careful study of 30 cases of fracture of the fibula through its

surgical neck in our series has shown the characteristics of a fracture by bending in all except 2 cases, in which it was clearly by torsion. When this lesion (by whatever mechanism) was unaccompanied by a diastasis of the inferior tibiofibular joint (or its equivalent, detachment of the anterior tubercle of the tibia), then the history has shown (in all but 1 case in which the history is unknown) either that the fracture was due to direct

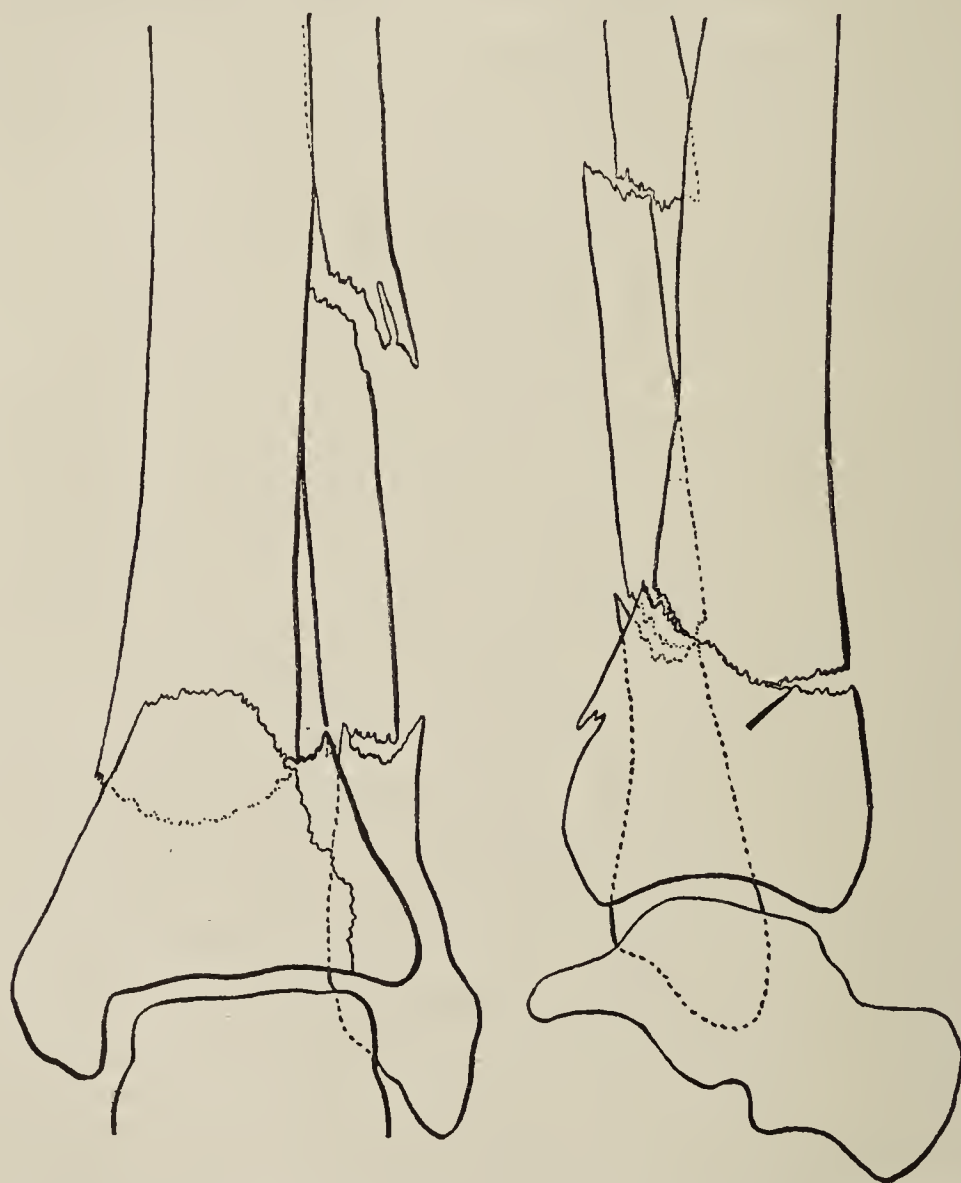


FIG. 48.—Comminuted supramalleolar fracture; probably by direct violence.

violence (Fig. 28) or that the clinical signs of a tibiofibular sprain were present though no diastasis was shown by the roentgen-ray. Quénu⁵⁵ (1912), however, for whose opinion every student of fractures has great respect, held that diastasis was not a necessary accompaniment of this type of fracture, though he acknowledged its extreme frequency, a fact to which he had called attention in 1909; and as early as 1907, he had

pointed out that this type of fracture was that which was most frequently accompanied by diastasis. But he was at that time (1907) inclined to the view that the fracture of the fibula occurred simultaneously with or even before the diastasis. But Destot⁵⁶ (1912) is firm in his belief that fracture of the fibula above the inferior tibiofibular joint demands as a preliminary a sprain or a diastasis of that joint. Is not then the legend to Fig. 67, p. 142, in his monograph (1911) inaccurate when it states that the diastasis shown was possible only because of the existence of a fracture near the head of the fibula?

Finally, as in fractures by outward rotation, the most advanced stage of abduction fractures may be regarded as one in which the entire lower end of the tibia is torn off as the representative of the internal malleolus; but in these also the fibula breaks characteristically by flexion through its surgical neck (Fig. 29)—it is not a mixed oblique fracture as in the third degree of fracture by external rotation already described (Figs. 18 and 19).

Diastasis and Lateral Displacements of the Foot. It is well to analyze more carefully what is meant by diastasis of the inferior tibiofibular joint.⁵⁷ It is easily seen in the prepared specimen that in all movements of the foot there is greater strain on the anterior than on the posterior inferior tibiofibular ligament; and that if the anterior ligament alone is divided, a separation of the fibula from the tibia to the extent of about 1 cm. becomes possible anteriorly, the fibula still being attached by the interosseous ligament and the posterior tibiofibular ligament. This degree of separation is sufficient to constitute a diastasis; lesser degrees, with incomplete rupture of the anterior tibiofibular ligament, and therefore without separation, constitute a sprain. But in many cases in which a temporary diastasis (disjunction) may have been present at the moment of the accident, it is no longer present when the patient comes under the surgeon's care or is sent for roentgenologic study; and at the latter time can only be presumed to have existed by certain signs, especially evidences of a sprain fracture or detachment of the anterior tubercle of the tibia (Fig. 30).

If, in addition to division of the anterior tibiofibular ligament, one divides also the feltlike interosseous ligament, then a separation of the fibula from the tibia almost to the distance of 3 cm. may be possible; but even with this amount of diastasis of the anterior border of the fibula from the tibia, these bones are still united by the posterior tibiofibular ligament, so that the fibula is not entirely free from the tibia though its lower end has become so movable as easily to permit a fracture by flexion.

Section of the posterior inferior tibiofibular ligament, as, indeed, noted by Quénu⁵⁸ (1907) permits only an insignificant separation of the fibula and tibia; and Quénu thinks that in life rupture of this ligament never occurs—at any rate it seems certain that this ligament is never ruptured alone, but only in conjunction with rupture of the anterior and interosseous ligaments. It is to be noted furthermore that it is next to an unheard of thing for the astragalus to be separated from the external malleolus, owing to the almost indestructible posterior band of the external lateral ligament; so that diastasis is not produced by a wedgelike action of the astragalus described by so many writers. I know of no case of true ascent of the astragalus between the tibia and fibula unaccompanied by the external malleolus: even in the beautiful case of diastasis recorded by Millikin,⁵⁹ though it is truly said that “the astragalus was jammed up between the outer surface of the tibia and the unfractured fibula” yet the astragalus had not been detached from the fibula and, therefore, cannot have acted as a wedge in driving the bones apart, though it certainly acted as a prop to keep them asunder. I have been at pains to reproduce this lesion on the cadaver, demonstrating that it is quite possible for the astragalus to become lodged against the outer surface of the tibia, between the latter bone and the fibula, without rupture of the posterior band of the external lateral ligament; though, of course, it is necessary to divide the internal lateral ligament and the inferior (anterior and posterior) tibiofibular ligaments, as well as the interosseous ligament and the interosseous membrane (the latter as far up as the upper third of the leg). And unless, in addition to the above, the anterior and middle bands of the external lateral ligament

were divided, the astragalus maintained itself only in very unstable equilibrium between tibia and fibula; though no doubt in life, the stability would be greater owing to muscular tension. Moreover, in the type of fracture with diastasis in which the fibular fragment is accompanied by a fragment detached from the external surface of the tibia (as in Cooper's illustration copied by Vidal de Cassis), and which is usually described as exhibiting ascent of the astragalus between the bones of the leg, or along the outer side of the tibia, the astragalus carries with it the external malleolus (the lower fibular fragment), so that in no true sense has the astragalus ascended between the bones. Of course, it is true that dislocation of the astragalus may occur, forward or backward, detaching it from the fibula; and there is no denying the possibility of its being dislocated upward between the intact tibia and fibula. All that I contend is that for it to become detached from the external malleolus in fractures of the leg bones at the ankle must be extremely rare, as there does not appear to be any such case on record.⁶⁰

The Posterior Marginal Fragment of the Tibia, and Posterior Displacement of the Foot. The existence of this fragment as the sole osseous lesion in a certain number of cases now on record,⁶¹ including one of our own, proves that it may be the earliest stage of a lesion involving the ankle-joint. But as there never has been any displacement of the fragment in these isolated lesions, it cannot be considered of much importance unless associated with a fracture of the fibula. As already noted, the existence of this fragment as a complicating lesion was well known to Cooper; it was noted by Earle⁶² (1828) and by Adams⁶³ (1835); it was observed at necropsy by Dupuytren "with surprise" according to Malgaigne⁶⁴ (1832); Thaon⁶⁵ presented a necropsy specimen and said he had often seen this fragment in experiments made by Tillaux; it was clearly and accurately if succinctly described by Nélaton,⁶⁶ (1847), and the fragment was recognized as a serious complication by Edmund Andrews⁶⁷ (1883, 1897). Since the introduction of the roentgen-ray, it has been studied by Chaput⁶⁸ (1899, 1907), Bondet²⁷ (1899), Grashey⁶⁹ (1907), Meissner⁶¹ (1907), Plagemann⁷⁰ (1911), Destot⁸ (1911), Quénu (1912-1915), and by

Stimson (in every edition of his book since 1899). Hence it was with surprise that surgeons who were tolerably familiar with the literature of their profession, as well as with fractures of the ankle, saw Cotton⁷¹ (1915) describe it as "a new type of ankle fracture" which "had never been adequately described in print and has apparently escaped the notice even of those who deal with fractures habitually;" and noticed his complacent comment that in certain circles it was called "Cotton's fracture;" as well as his statement that he believed there were no necropsy specimens.⁷² Cotton, however, did well to call attention to its frequency; as did Speed in a paper which was not published until after Cotton's paper was read, though it appeared in print before the latter.

The mechanism by which this fracture is produced is almost certainly, as contended by Lucas-Championnière,⁷³ a crushing force from below upward;⁷⁴ it is possible that traction by the posterior inferior tibiofibular ligament, through the medium of the fractured lower end of the fibula, may aid in displacing the fragment, even if it cannot be the sole cause of its detachment. The size of the fragment varies from a small portion of the lip to a large fragment, extending 10 cm. up the posterior surface of the shaft. McKnight, at the meeting of the Philadelphia Academy of Surgery, May 2, 1921, showed a roentgenogram (anterioposterior view only) of a fracture which I believe conformed to this type, though the fragment was the largest with which I am acquainted: the fragment (an isolated lesion, without displacement) included nearly all the posterior lip of the tibia, as well as its entire lateral (fibular) border; and the apex of the large wedge extended to a point about 10 cm. above the articular surface, on the posterolateral border of the tibia.

The lesion corresponds, as Tanton²⁹ (loc. cit., p. 171) has noted, to Rhea Barton's fracture of the posterior margin of the radius.

It is a much more frequent complication than commonly supposed. Among our 300 cases it was present no less than fifty-eight times, or in 19 per cent of all the cases; and in 51, or 50 per cent, of those conforming to the "low Dupuytren" type.

TABLE I.—INCIDENCE OF POSTERIOR MARGINAL FRACTURES

	Total ankle fractures	Posterior marginal fractures.			
		Associated.	Isolated.	Total.	Per cent.
Ashhurst and Bromer . . .	300	57	1	58	19.0
Chaput (Les fractures mal-léolaires, Paris, 1907) . .	136	42	0	42	30.0
Destot (Quénu: Bull. et mém. Soc. de chir. de Paris, 1913, xxxix, 165)	1700	139	6	145	8.5
Quénu (Rev. de chir., 1912, xlv, 260)	129	11	1	12	9.3
Sear (Med. Jour. Australia, 1917, i, 526)	156	26	3	29	18.6
Speed (Surg., Gynec. and Obst., 1914, xix, 73) . . .	161	16	0	16	10.0

Incidence of Associated Posterior Marginal Fractures

Type.	Ashhurst and Bromer		Destot	
	No.	Per cent.	No.	Per cent.
Fibula, oblique, mixed	0	..	0	
Low Dupuytren	51	88.0	89	64
Bimalleolar	0	..	17	12
Pott's	5	8.6	29	20
Adduction	1	1.7	0	
Maisonneuve	0	..	4	3
	57		139	

There is sometimes confusion between this posterior marginal fragment and the intermediate fragment ("third fragment of Tillaux"), as remarked before.²³ The posterior tubercle of the tibia, which limits posteriorly the gutter for the reception of the fibula (Fig. 4), and to which is attached the posterior tibiofibular ligament, may be fractured by the pull of this ligament. It appears then in lateral roentgenograms as an infraction or sprain fracture, but does not involve the ankle-joint. In the true marginal fractures, on the other hand, the line of fracture always extends into the tibial plafond; the fragment (contrary to what is said by Tanton) usually remains attached to the external malleolus by the posterior tibiofibular ligament, and is often displaced backward with the external malleolus; sometimes (more frequently than thought) it may be detected in anteroposterior roentgenograms as a deltoid fragment; and, if large, its shadow sometimes overlaps that of the median border of the tibia, giving a double contour.

The posterior marginal fragment, as already remarked, may

occur as an isolated lesion; only once in our series (Fig. 44). It may be associated with (*a*) merely a diastasis, with sprain fracture of the anterior tubercle of the tibia (Fig. 31); (*b*) it may accompany the mixed oblique fracture of the fibula in the various stages of the fracture by outward rotation, being small (Fig. 32), medium sized (Fig. 33), or large (Fig. 34); and (*c*) it occurs also with fractures by abduction (Figs. 35 and 36), though very much less frequently than with external rotation fractures. Quénu has had one case in which fracture of the internal malleolus was the only other lesion, and another fracture of precisely the same type has come under our notice since our series of 300 cases was completed.

Its presence is not necessary to permit the occurrence of posterior displacement of the foot (Figs. 16 and 25), but certainly favors it. This posterior displacement is very rare without the posterior marginal fragment. Either a posterior marginal fragment must exist, or there must be rupture of the posterior inferior tibiofibular ligament. It is only a continuance of the force (now the weight of the body) after the fracture has been produced, that causes the displacement, since there are in our series no less than fourteen instances (out of a total of fifty-eight posterior marginal fractures) with slight if any displacement.

The factors which permit posterior displacement of the foot deserve a few words. Quénu was the first, I believe, to point out that the essential lesion is freeing the lower end of the fibula from the tibia; and I may remark that this necessity is merely a corollary of what has been insisted on so often in these pages, namely, the indissolubility of the union between the astragalus and the fibula. I cut everything else at the ankle (tendons and ligaments as well as all other soft parts) leaving only the middle and posterior bands of the external lateral ligament attaching the external malleolus to the foot. Under these circumstances even an incomplete posterior dislocation of the foot cannot occur (except by rotation of the foot inward around the long axis of the leg, a mechanism which does not occur in life). I then cut the middle band of the external lateral ligament; but still no posterior displacement could be produced. Next I fractured the fibula 7.5 cm. (3 inches)



FIG. 49.—Anteroposterior view of ankle-joint. (See page 328.)



FIG. 50.—Lateral view, plantar flexion.



FIG. 51.—Lateral view, dorsiflexion.



FIG. 52.—Rotation outward, obliterating outline of the posterior tubercle. (Same happens in mesial deviation of the tube.) The anterior tubercle overlaps the lateral margin of the fibula.



FIG. 53.—Rotation inward; very little change from the normal. (Same happens in lateral deviation of the tube.)

above its tip; but this did not permit posterior displacement. Finally, I divided the anterior inferior tibiofibular ligament, and even the interosseous ligament; but so long as the fibula remained attached to the tibia, and the astragalus to the fibula, no posterior dislocation could be produced. On another foot, I divided all structures uniting the foot to the fibula except the middle band of the external lateral ligament; but no dislocation of the foot was possible; then I divided also the posterior inferior tibiofibular ligament and the interosseous ligament (which allowed less diastasis than when the anterior tibiofibular ligament was divided), but still no posterior dislocation of the foot was possible except by rotating it inward around the one remaining ligament as a pivot.

As a result of these investigations, it may be concluded that either the middle or the posterior band of the external lateral ligament is sufficient to hold the astragalus against the external malleolus, and that (even after fracture of the internal malleolus or rupture of the internal lateral ligament) *no backward dislocation of the foot can occur* (1) unless the astragalus is freed from the external malleolus by rupture of both the middle and posterior bands of the external lateral ligament (a lesion which apparently has not been recorded in association with fractures at the ankle); (2) or unless the external malleolus is freed from the tibia (*a*) by diastasis of the inferior tibiofibular joint, with or without detachment from the tibia of an intermediate fragment; (*b*) by fracture of the fibula in such a way as to detach with the lower fragment of the fibula the fibular insertions of both the middle and posterior bands of the external lateral ligament (in other words, unless a virtual diastasis occurs, but one in which the intermediate fragment belongs to the fibula instead of to the tibia). One very frequent fracture of the fibula (the mixed oblique of Destot) fulfils these requirements; as does another less usual, namely a transverse fracture of the external malleolus proper below the level of the tibia. But as the latter fracture usually is subperiosteal or incomplete, and without displacement, it follows that posterior displacement of the foot with this lesion is unknown.

Therefore, the fractures which may be accompanied by posterior displacements of the foot are those of the Pott or

Dupuytren type in which diastasis is the rule; and those of the low Dupuytren variety, in which the fracture of the fibula is of the mixed oblique type. Quénu⁷⁵ is wrong, I am sure, in claiming (1912) that the existence of a posterior marginal fragment is a necessary condition for the occurrence of a posterior subluxation (Figs. 16 and 25); but it is well to remember that the distorted shadows of a roentgenogram in a case of fracture of the low Dupuytren variety, in any of its stages, may mislead the observer into the belief that a posterior displacement is present, when the appearances are due entirely to an outward rotation of the astragalus around the long axis of the leg.

Forced Movements of Adduction (Tibial Flexion). Since the time of Cooper and that of Maisonneuve and Bonnet, there has been little dispute about the mechanism of these fractures: it has been generally recognized that a tearing off of the external malleolus is the first lesion (Fig. 37), followed by a compression fracture of the internal malleolus (Figs. 38 and 39); or, when the weight of the body forces the tibia heavily on the displaced astragalus, a splitting upward of the tibial shaft occurs, the line of fracture commencing at some point of the articular surface, splitting this in the sagittal plane and terminating on the median border of the tibia at a variable distance above the internal malleolus (Figs. 40, 41 and 42). The more nearly longitudinal the line of fracture, the less necessary will it be for the external malleolus to be fractured as a preliminary step: such fractures verge into those due to comminution upward in the long axis of the limb. Stimson⁷⁶ (1912) gives an admirable illustration of such a longitudinal fracture of the tibia without any lesion of the fibula. But if the displacement in these longitudinal fractures is marked enough, the fibula may break secondarily (by flexion or torsion) above the inferior tibiofibular joint, as the lower end of the fibula is carried inward with the astragalus (Fig. 41). A variant of this type is the lesion recorded by Silhol⁷⁷ in which the fibular fracture is replaced by an intermediate fragment detached from the tibia. Even in one of our own cases (Fig. 40), there is an intermediate fragment, in addition to the fracture of the fibula.

The supramalleolar fracture by adduction, produced experimentally by Tillaux, and already mentioned at page 279, may be considered the most advanced degree of this type (Figs. 42 and 43.)

SUMMARY. These then are the abnormal movements—external rotation, abduction, adduction—which are responsible for the great majority (95 per cent) of fractures about the ankle: external rotation causes about 61 per cent, abduction about 21 per cent, and adduction about 13 per cent of the lesions. The remaining small proportion (5 per cent) consists chiefly of those fractures which may be recognized either as caused by compression in the long axis of the limb, or by direct crushes; or even by very rare forced movements such as straight flexion or extension, internal rotation, etc. The fractures by compression in the long axis of the limb include (a) the isolated fractures of the tibial margins (anterior, posterior (Fig. 44), median or even lateral), which Sear⁷⁸ speaks of as “vertical plane fractures;” (b) comminuted fractures of the tibial plafond (Fig. 45), and (c) T- or Y-fractures involving the ankle-joint (Figs. 46 and 47), which may be regarded as an advanced degree of the supramalleolar V-fractures described by Gosselin,⁷⁹ the latter usually being complicated by a fissure extending into the ankle-joint, the mechanism being the same as in those of the radius with comminution of the wrist fragment.⁸⁰ The supramalleolar fractures of Malgaigne⁸¹ (1847) are thus distributed according to their mechanism, some into those by adduction, others as due to compression in the long axis of the limb, and a few probably due to direct violence (Fig. 48). We count no supramalleolar fracture as one involving the ankle unless it falls within 4 cm. of the joint level (Richet, 1875). Fractures as close to the ankle-joint as 4 cm. compromise its functions as surely as do supracondylar fractures of the humerus compromise those of the elbow.

Now, it is because of the impossibility of classifying together anatomically lesions which, as Stimson says, are merely alternative, or whose differences are due to the early cessation of the force before the typical form has been reached; or, I may add, to its continuance after the typical stage has been passed—it is because of this impossibility, I repeat, that I

believe a classification based on mechanism, imperfect though it be, is, nevertheless, more easily understood and remembered. And it is for this purpose that Dr. Bromer and I have ventured to arrange our series of fractures as shown in Table II. Our aim has been to place under each mechanism, first, the simplest resulting form of fracture, and to advance thence to more

TABLE II.—CLASSIFICATION OF THREE HUNDRED ANKLE FEACTURES

A. Fractures by External Rotation—	
1. First Degree: Lower end of fibula only ("mixed oblique")	79 (26.00%)
2. Second Degree: Same, <i>plus</i> rupture of internal lateral ligament or fracture of internal malleolus ("low Dupuytren")	100 (33.00%)
Viz.,	
(a) Internal lateral ligament, uncomplicated . . .	13
Internal lateral ligament complicated by posterior marginal fragment of tibia	13
(b) Internal malleolus, uncomplicated	32
Internal malleolus complicated by posterior marginal fragment of tibia	42
3. Third Degree: Same, <i>plus</i> fracture of whole lower end of tibia, representing the internal malleolus	5 (1.70%)
<hr/>	
Total fractures by external rotation	184 (61.00%)
B. Fractures by Abduction (Fibular Flexion)—	
1. First Degree: Internal malleolus only	20 (6.60%)
2. Second Degree: Same <i>plus</i> fracture of fibula (transverse, above or below, tibiofibular joint)	41 (13.70%)
(a) Below inferior tibiofibular joint (no diastasis) ("bimalleolar fracture")	13
(b) Above inferior tibiofibular joint (with diastasis) ("Pott's fracture," "Dupuytren type")	28
3. Third Degree: Internal malleolus represented by whole lower end of tibia	2 (0.66%)
<hr/>	
Total fractures by abduction	63 (21.00%)
C. Fractures by Adduction (Tibial Flexion)—	
1. First Degree: External malleolus only, transverse, at or below level of tibial plafond	27 (9.00%)
2. Second Degree: Same, <i>plus</i>	
(a) Internal malleolus below level of tibial plafond ("bimalleolar fracture")	3
(b) Median surface of tibia up and in from joint surface	8
3. Third Degree: Same, <i>plus</i> whole lower end of tibia ("supramalleolar fracture by adduction")	2 (0.66%)
<hr/>	
Total fractures by adduction	40 (13.30%)
D. Fractures by Compression in Long Axis of Leg—	
1. Isolated marginal fractures	1
2. Comminution of tibial plafond	3
3. T- or Y-fractures ("V-fractures of Gosselin")	4
<hr/>	
Total fractures by compression in long axis of leg	8 (2.70%)
E. Fractures by Direct Violence (Supramalleolar Types) . . .	5 (1.70%)

complicated lesions, noting at the same time in their appropriate places the variants and the complications of the simple or the more complex lesions which were encountered. It is true that it is not always easy to determine the mechanism, even with all the aid derived from the clinical history, the roentgen-ray and a knowledge of the lesions which can be produced on the cadaver; but the more one studies the subject, the fewer exceptions he will find to the general laws of mechanics; and the more his experience increases, the easier will it become to recognize the variants from the typical lesions. Those fractures which have given us most concern are the true bimalleolar fractures with little or no displacement, since these may be caused possibly by external rotation, certainly both by abduction and adduction. But as the total number of these fractures observed is small (16 cases or only 5 per cent of the entire series), and as even among this number

TABLE III.—ANATOMOPATHOLOGIC CLASSIFICATION OF THREE HUNDRED ANKLE FRACTURES

A. Fibula, Below Inferior Tibiofibular Joint—			
1. Alone (slight or no displacement, often subperiosteal)	27 (9.00%)		
2. Same, <i>plus</i> internal malleolus below tibial plafond (includes bimalleolar fractures by adduction and by abduction)	16 (5.00%)		
3. Same, <i>plus</i> split of tibia up and in	8 (2.60%)	51 (17.0%)	
B. Fibula, Obliquely Through Inferior Tibiofibular Joint—			
1. Alone (slight or no displacement, often subperiosteal)	79 (26.00%)		
2. Same, <i>plus</i> rupture of internal lateral ligament	26 (9.00%)		
3. Same, <i>plus</i> fracture of internal malleolus	74 (25.00%)	179 (60.0%)	
C. Fibula, Above Inferior Tibiofibular Joint—			
1. Alone (slight or no displacement, often subperiosteal)	0		
2. Same, <i>plus</i> rupture of internal lateral ligament and diastasis	8 (2.70%)		
3. Same, <i>plus</i> fracture of internal malleolus and diastasis	20 (6.60%)	28 (9.3%)	
D. Tibia, Involving Ankle-joint—			
1. Internal malleolus alone (slight or no displacement, often subperiosteal)	20 (6.60%)		
2. Isolated marginal fractures	1 (0.33%)		
3. Comminuted T- and Y- fractures	7 (2.30%)	28 (9.3%)	
E. Supramalleolar Fractures, Not Involving Ankle-joint Directly			
	14 (4.30%)	14 (4.3%)	

the mechanism was reasonably certain in all but 10 cases (3 per cent of the entire series), the margin of error is small.

TABLE IV.—CATALOGUE OF THREE HUNDRED FRACTURES INVOLVING THE ANKLE

	No. Cases.
A. By Rotation of the Astragalus Outward Around the Long Axis of the Leg—	
I. First Degree: Lower end of fibula only. Oblique or spiral fracture from above and behind, down and front, the line of fracture passing through the inferior tibiofibular joint. Line varies from nearly transverse to nearly longitudinal, but is always higher on posterior than on anterior surface of fibula. In 84 per cent of cases the lower anterior end of line of fracture was on anterior surface of external malleolus between its tip and the anterior inferior tubercle of the tibia; in 8 per cent of cases it was above the anterior tubercle (when there was diastasis or the tubercle was detached); and in 8 per cent it passed through the fibula at the level of the anterior tubercle on the tibia. Slight or no displacement	76
<i>Variant:</i> Fracture of fibula in <i>upper third</i> from lesion at ankle (Maisonneuve, 1840)	1
(In this case there was incomplete fracture of fibula below its head (Fig. 17) but without bony lesion at ankle.)	
<i>Complication:</i> Intermediate fragment	2
	— 79
II. Second Degree: Fibula as in first degree, <i>plus</i> rupture of internal lateral ligament or fracture of internal malleolus. There may be slight or considerable lateral displacement; but in uncomplicated cases there is rarely a real posterior displacement, the apparent posterior displacement seen in lateral roentgenographic views being due usually to outward rotation which distorts the shadows.	
(a) Internal lateral ligament (in roentgenograms this lesion is differentiated from first degree only by presence of lateral displacement of astragalus from internal malleolus with slight displacement of lower fibular fragment)	12
<i>Variant:</i> Diastasis without fracture of fibula (Fig. 16)	1
<i>First Complication:</i> Fracture of posterior tibial margin. Line of fracture usually extends about 2 cm. up along posterior surface of tibia, and running down thence nearly vertically, detaches a mere chip or more often one-fourth to one-third of the articular surface; rarely as much as one-half	10
<i>Variant:</i> Diastasis without fracture of fibula, but with posterior marginal fragment (Fig. 31)	1
<i>Second Complication:</i> Intermediate fragment	0
<i>Third Complication:</i> Second fracture of fibula in upper third (Maisonneuve's fracture; these two cases occurred in opposite legs of same patient)	2
(b) Internal malleolus	32
(With slight or no displacement, 20 cases.)	
<i>First Complication:</i> Fracture of posterior tibial margin (Figs. 32, 33 and 34)	40
(Slight or no displacement, 14 cases.)	
(Lateral and posterior displacement, 26 cases.)	
<i>Second Complication:</i> Intermediate fragment	2
	— 100

TABLE IV.—CATALOGUE OF THREE HUNDRED FRACTURES INVOLVING THE ANKLE.—(Continued)

	No. Cases.
III. Third Degree: Internal malleolus represented by whole lower end of tibia	5
(This will include most cases of separation of lower epiphysis of tibia.)	
Total fractures by mechanism of outward rotation. .	184
B. By Abduction (Fibular Flexion) of Foot—	
I. First Degree: Fracture of internal malleolus only, transverse, at or below level of tibial plafond. No displacement . .	19
II. Second Degree: Rupture of internal lateral ligament or fracture of internal malleolus, as in first degree, followed by fracture of fibula more or less transverse either below or above inferior tibiofibular joint	
(a) Fracture of fibula below inferior tibiofibular joint (<i>i. e.</i> , of external malleolus proper) with fracture of internal malleolus or rupture of internal lateral ligament . .	13
(with no displacement, 3 cases; internal malleolus displaced more than external, 4 cases; both malleoli equally displaced, 6 cases)	
Variant: Crush of calcaneum (replacing fracture of internal malleolus) followed by compression fracture of external malleolus	1
(b) Fracture of fibula above inferior tibiofibular joint (in lower third), the line of fracture indicating a fracture by flexion (<i>Biegungsbruch</i>), with slight or no torsion; slightly oblique, often comminuted, with detachment of a wedge-shaped fragment from the surface of flexion, and its apex toward the surface of extension .	28
	— 42
1. Fracture of fibula alone, or with rupture of internal lateral ligament	6
Variant: Diastasis without fracture of fibula	2
2. Fracture of fibula with fracture of internal malleolus at or below level of tibial plafond . .	12
First Complication: Posterior marginal fragment of tibia (Figs. 35 and 36)	5
Second Complication: Intermediate fragment	3
III. Third Degree: Internal malleolus represented by whole lower end of tibia (this will include some cases of separation of lower epiphysis of tibia)	2
Total fractures by mechanism of abduction	63
C. By Adduction (Tibial Flexion) of Foot—	
I. First Degree: Fracture of external malleolus, transverse, at or below level of tibial plafond. Slight or no displacement, often subperiosteal	26
Complication: Posterior marginal fragment of tibia (infract-ion)	1
	— 27
II. Second Degree: Fracture of external malleolus as in first degree, <i>plus</i> :	
(a) Fracture of internal malleolus below level of tibial plafond	2
Variant: Fracture of external malleolus represented by rupture of external lateral ligament	1
(b) Fracture of median surface of tibia up from tibial plafond	8
	— 11

TABLE IV.—CATALOGUE OF THREE HUNDRED FRACTURES INVOLVING
THE ANKLE.—(*Continued*)

	No. Cases.
III. Third Degree: Fracture of fibula usually above inferior tibio-fibular joint, <i>plus</i> fracture across tibia above ankle-joint (Tillaux, 1872)	2
Total fractures by mechanism of adduction	40
D. By Compression in Long Axis of Leg—	
I. Isolated marginal fractures of tibia: anterior margin, none; posterior margin, 1; median margin, none; lateral margin, none	1
II. Comminution of tibial plafond	3
III. T- or Y-fractures of tibia into ankle-joint	4
	8
E. Fractures by Direct Violence (Supramalleolar Types)	5
Grand total	300

It will be noted that in our classification the fibular lesion dominates the clinical picture in the first two classes (those of outward rotation and abduction), and that these correspond to the first grand division of Destot's classification—those fractures in which the equilibrium of the foot is involved; while the third, fourth and fifth classes correspond to Destot's second division—those fractures which involve the tibial pestle and compromise the function of support. For the sake of completeness we also give an anatomopathologic classification, constructed on the same principles (Table III).

Following the classifications is a catalogue of the lesions encountered in this series of cases, which, with the aid of the classifications as a guide, we hope may prove of interest to students of fractures (Table IV).

ROENTGEN-RAY STUDY OF THE ANKLE-JOINT

BY DR. BROMER

I. THE NORMAL ANKLE-JOINT—ANATOMY

By means of the roentgen-ray, the structure of the ankle-joint can be most satisfactorily demonstrated. The tibio-fibular mortise is shown (Fig. 49), and below it the trochlea of the astragalus fitting into it as a tenon. The "plafond" and the "cheeks" of the mortise are easily recognized, likewise the

longer external malleolus and the anterior and posterior tubercles of the fibular groove. In the lateral view (Fig. 50), the low projection of the posterior lip, called by Destot the posterior malleolus, reinforcing the mortise posteriorly, can easily be visualized; and the point where it meets the trochlear surface of the astragalus, acting as a check on the latter when the foot is in plantar flexion as in walking, is most noticeable. (The degree of plantar flexion in the roentgenogram is much greater than is assumed in walking, but is used more fully to demonstrate the check.) In this view, the longer external malleolus is shown overlapping the shadow of the internal malleolus. With stereoscopic plates they usually can be fairly well distinguished. The lateral tubercle on the posterior surface of the astragalus (*os trigonum*) is the farthest posterior shadow of the astragalus. In the interpretation of roentgenograms of the ankle-joint, due care must be exercised not to diagnose a sprain-fracture of the posterior tubercle, where an accessory bone, the *os trigonum*, often is found. In general, no fracture exists, and it is this bone that is present if the surfaces of the fragment are smooth and rounded.

II. FUNCTION AND MOVEMENTS OF THE ANKLE-JOINT

The roentgen-ray can be used to demonstrate the movements of the ankle-joint. Thus the rotation of the astragalus about the axis previously described by Dr. Ashhurst in full dorsiflexion and plantar flexion is shown in Figs. 50 and 51. The movements of the fibula were shown in the following way: A normal ankle was examined; the ankle and leg of the subject being securely bound to the table, a Bowen stereoscopic plate holder was placed beneath the ankle-joint. Lateral views were made first with the foot in full dorsiflexion, then later with the foot in full plantar flexion. Great care was exercised that the position of the leg—*i. e.*, tibia and fibula—was not altered. The stereoscopic plate holder gave an exact duplicate position for the second exposure. The tube was in no way shifted. Anteroposterior views were taken in the same way. In this plane, the heel was allowed to project as far as possible over the edge of the plate holder in order to obviate any change

in height of the tibia above the plate due to change of position of the os calcis. The superior tibiofibular articulation was examined in the same way.

It was found that these views could be superimposed over diffused strong light. As an additional check the distances between the same relative points were accurately measured. The fact that the images of the tibia could be accurately superimposed would tend to rule out any possibility of error due to change in height of tibia. It was found that in full plantar flexion in the anteroposterior view the fibula moved inward and downward 1 mm. or, *vice versa*, so much expansion and movement upward occurred in full dorsiflexion. No change of the fibula in relation to the tibia could be found in the lateral views.

With regard to the upper extremity of the fibula, the forward movement of the fibula in plantar flexion was shown in the lateral view by an increase of 1 mm. in the distance between the posterior lip of the upper extremity of the fibula and the posterior border of the tibia. In the same way in the anteroposterior view the head of the fibula was found lying farther in toward the midline with the foot in plantar flexion than it was with the foot in dorsiflexion. All of these measurements were made by means of calipers, and the same level was obtained by means of superposition of the films over diffused strong light.

III. SOME DIFFICULTIES ENCOUNTERED IN INTERPRETATION OF THE ROENTGENOGRAMS OF THREE HUNDRED CASES

During the intensive study of the roentgenograms, from the point of view of roentgen interpretation, most interesting questions arose, necessitating definite solutions before any classifications or any theories of mechanism could be determined. These questions came almost entirely from a lack of standardized technic. The 300 cases had been examined by four different roentgenologists. The technic of only one of them was known. Immediately, the question arose: Does not this, or that, represent some variation from the real normal,

produced by variations in the procedures of these different roentgenologists, rather than a pathologic condition?

Textbooks on roentgenology describe certain more or less standardized positions for examination of the ankle-joint. They point out anatomic landmarks above which to center the target of the tube. But none of them describe the variations in shadow which may occur: for instance, from increased or decreased target plate distance, or from variations produced by lateral shifting of the target, or by variations in posture of the joint. It would seem that opportunities for error are numerous. The very nature of hospital dressings—so different in the various institutions—constitutes one of the most prolific sources of error. The difficulty of exact centering through a plaster-or-Paris dressing or of an ankle encased in a fracture box is obvious.

The discussion which follows may seem to be far-fetched to some and futile to others. I am well aware of the conditions under which many busy roentgenologists work. I realize that a plate or film placed under an ankle with the target at any given distance above it, and the perpendicular assumed or guessed at, with a proper exposure technic will result in a so-called excellent negative. We admit that in a busy hospital service most of our patients were so examined. We admit that to the best of our knowledge no patients in any way afterward suffered, clinically, from such unstandardized examination at our hands. It is entirely probable that many clinicians can and will say, Why more? But I contend that in our study of these cases we were greatly hampered by such methods in arriving at definite conclusions. Gross lesions are usually apparent and easily diagnosed. The fine points in diagnosis are the difficult ones. It is only by exact methods that the science of diagnosis is advanced and the sum total of our knowledge of the subject increased.

The first difficulty that arose, the solution of which was of utmost importance, was the exact determination of just what on the roentgenogram determines diastasis of the lower tibio-fibular junction. The normal ankle-joint was roentgenographed under all possible conditions, in an effort to study the possible variations in shadows of the normal due to changes

in technic. It was first examined at various target plate distances with the foot and target in the perpendicular plane; then the same ankle was roentgenographed with the leg in different angles of rotation and also with the foot in full dorsiflexion and plantar flexion, and again with the target deviated to either side from the perpendicular plane. A study of these results shows the necessity for the roentgenologist's deciding on one target plate distance for all examinations, also for him to formulate or design some scheme for quickly securing in each case the same perpendicular plane of the ankle and target, especially in the anteroposterior view. A long upright rod with another at right angles, attached to its lower end, can readily be made and used for this purpose (goniometer).

From the results obtained on the roentgenograms showing the variations in target plate distance, it can be assumed that shadows on roentgenograms follow certain rules. Thus, as this distance decreases, all shadows proportionately increase provided there is no shift of the target in any direction, or any change in the position of the ankle, as, for instance, rotation of the limb. Thus the distance between the shadow of the line of the posterior tubercle on the fibular groove and that of the mesial margin of the fibula is increased if the target is moved nearer the plate, but likewise the width of the fibula and tibia seems to be proportionately increased. However, by no possible manipulation of the tube to either side up to 75 degrees from the perpendicular, or by inward or outward rotation of the leg to 80 degrees can the shadow of the most lateral point of the anterior tubercle be made to pass to the median side of the shadow of the median border of the fibula. (It seemed that the above-mentioned angles were the limits of the possible errors that could have been made in the roentgenograms of our series.) Hence it was felt that we could safely say that whenever this did happen, tibiofibular diastasis was established. In fact, if the space between the lateral margin of the fibula and the lateral border of the anterior tubercle exceeds more than two thirds of the width of the fibula, it is most probable that the first degree of diastasis (Fig. 36) exists. This would seem to be certainly the case if the roentgenogram was made with the foot and ankle and tube in the absolute perpendicular plane (Figs. 49, 52 and 53).

Chaput³⁹ (1912) gave figures in millimeters showing variations of the "clear space" as he called it between the line of the posterior tubercle and the mesial border of the fibula, and claimed that if this space exceeded more than 3 mm., or if the area of the overlapping shadows of fibula and anterior tibial tubercle was less than 10 mm., diastasis was present. The objection to this as compared with the above method is obvious. Measurements on the roentgenogram vary greatly with changes in technic, target plate distance, etc., with age periods and in the different sexes. So a method establishing a means of estimation proportionate for each individual case is manifestly the best.

The larger the clear space between these bones, the greater the degree of diastasis. If there is additional roentgen-ray evidence of a sprain-fracture or detachment of the anterior tubercle, there is double assurance (Figs. 30 and 31). But here again one must make due allowance for the fact that when the patient reaches the roentgenologist reduction often has been established. The clear space will then not be abnormally wide, and diastasis can only be presumed to have existed by the evidence of a sprain-fracture or a fracture of the anterior tubercle. If these are not present, then no diagnosis of diastasis can be made by the roentgen-ray. It seems possible that by a very complete study of normal ankles in both sexes, and in all age periods under identical conditions of technic, more absolute criteria can be established whereby this diagnosis can be made.

The clear space between the internal malleolus and the astragalus in the anteroposterior view is also a most interesting study. What determines the earliest or first degree of lateral displacement of the astragalus? This certainly is of the utmost importance in the determination of such displacement, particularly the so-called outward displacement. It was found that a 10 degree inward rotation of the leg increased this clear space, that the same amount of external rotation correspondingly decreased it; that variations in target plate distance proportionately increased or decreased it as the case might be, that tibial and fibular flexion of the foot both decreased it. So if a minute, careful diagnosis is to be made, the absolute

perpendicular must be maintained. Had we had all roentgenograms of the 300 cases studied, made under identical technic, such diagnosis could have been made. We feel that quite a considerable degree of displacement had to be present, in fact so much that it would be apparent under any condition, any position of tube or ankle, before we could say definitely in the foregoing cases that a lateral dislocation existed. Probably also a better idea of rupture of the internal lateral ligament can be obtained by means of this clear space. Thus, when the internal malleolus is intact in the pure abduction fractures, this space may afford an idea of whether or not the internal lateral ligament has ruptured. Here again faulty methods prevented exact conclusions, and this again emphasizes the possibility of study under standardized conditions.

Study of the fibular groove led to the definite conclusion that the anterior tubercle always casts the more lateral shadow, the posterior tubercle the more mesial. This was confirmed by placing pieces of lead of different sizes on the lips of the groove in a living subject and then roentgenographing the ankle. It was noted, in the negatives made with deviations of the tube and change in position of the ankle, above described, that in the perpendicular plane the line of the posterior tubercle was always clearly defined, that this clear definition persisted when the leg was rotated in or with lateral deviation of the target; but with the leg rotated out or with mesial deviation of the target, this line had a tendency to disappear, becoming merged with the dense shadow of the thicker middle portions of the tibia. It is apparent that such variations may materially interfere with the exact determination of just what constitutes the so-called posterior fragment. It may also interfere in the same way with recognition of the intermediate fragment. If this line remains intact, the posterior tubercle is not injured and the posterior inferior tibiofibular ligament is also probably not affected. It is possible that a deltoid shaped posterior marginal fragment broken from the tibia at a point mesial to and behind the posterior tubercle may be so displaced that its shadow overlaps the line of the posterior tubercle. The exact portion fractured can then only be determined by means of stereoscopic plates, and even then this

is sometimes impossible. A fracture with such displacement was produced on a dried specimen, likewise another involving the posterior tubercle so as to interfere with the line of the latter on the roentgen-ray negative. In the anteroposterior view, they were indistinguishable, both appearing to be fractures of the posterior tubercle. However, stereoscopic films showed quite plainly the character of the first, *i. e.*, a fragment displaced behind the tubercle and really not involving it.

In conclusion, this study has shown that while the roentgen diagnosis of the gross lesions of the ankle-joint is a comparatively easy matter for the trained roentgenologist, the finer points of diagnosis so necessary to a thorough understanding of the mechanism are entirely dependent on more exact methods of technic than are often now employed.⁸²

REFERENCES

1. Pott: Some Few General Remarks on Fractures and Dislocations, London, 1769, p. 57.
2. Dupuytren: Ann. méd.-chir. d. hôp. et hosp. civ. de Paris, 1819, p. 1.
3. Cooper: Treatise on Dislocations and Fractures of the Joints, London, 1822.
4. Maisonneuve: Arch. gén. de méd., 1840, i, 165, 433.
5. Tillaux, cited by Gosselin: Bull. de l'Acad. de méd., Paris, Series 2, 1872, i, 817.
6. Hönigschmied: Deutsch. Ztschr. f. Chir., 1877, viii, 239.
7. Stimson: New York Med. Jour., 1892, lv, 701.
8. Destot: Traumatismes du pied et rayons-x, Paris, 1911.
9. It should be borne in mind that Pott is discussing the importance of treating fractures of the leg bones in the flexed position (the limb resting on its outer surface with the knee bent), and hence that he speaks of fractures of the fibula only incidentally.
10. Bazille: Mém. sur les sujets proposés pour le prix de l'Acad. Roy. de Chir., Paris, 1778, iv, 563.
11. Bromfield: Chirurgical Observations and Cases, London, 1773, ii, 78.
12. Pouteau: Oeuvres posthumes, Paris, 1783, ii, 267.
13. This memoir was said by Nélaton (Eléments de path. chir., Paris, 1844, i, 810) to have been read by Dupuytren, in 1813, before the Académie des sciences. However, I have searched the "Procès-verbaux des séances de l'Académie des sciences" (Paris, vol. v, 1812-1815) and find no reference to such an event; and Quénu (Rev. de chir., 1912, xlv, 5²) states that he has had the original records of the Academy searched, page by page, without finding any trace of Dupuytren's Memoir; and says that "until further information" he will consider as "mémoire princeps" the appearance of the essay in the "Annuaire médico-chirurgicale des hôpitaux et hospices civiles de Paris," 1819, p. 1. The essay is most easily accessible where first reprinted, in the second edition of Dupuytren's "Leçons orales" (Paris, 1839, i, 275).
14. Dupuytren: Leçons orales de clinique chir., éd. 2^e, Paris, 1839, i, 328.
15. Quénu: Rev. de chir., 1912, xlvi, 367.²

16. Hutchinson: *Trans. Path. Soc., London*, 1887-1888, xxxix, 238.
17. Dupuytren:¹⁴ p. 368.
18. Though unfortunately, it is not known by his name, which is attached to a fracture produced experimentally by him on the cadaver, but which he never saw clinically. The most frequent and most typical fracture the French now call the "low Dupuytren," to distinguish it from the true Dupuytren or Pott fracture, which they name "Dupuytren type."
19. This is the "intermediate fragment" known to the French as the "third fragment of Tillaux," who produced it frequently in his cadaveric experiments.
20. Richet (*Unión méd.*, 1875, xx, 142) described such a case in which the astragalus was penetrated by the upper fragment of the fibula. Roentgenograms in anteroposterior view often show such an appearance, but the lateral views I have seen have always demonstrated that the fibula was behind, or very rarely in front of, the astragalus. Richet's lesion was demonstrated at necropsy.
21. Vidal de Cassis: *Traité de pathologie externe*, 2d ed., Paris, 1846, ii, 394.
22. Dupuytren contended that the usual fracture which he described (and which he thought corresponded to that shown in Pott's illustration) was produced by a primary tearing off of the external malleolus when the foot turned so that the patient stepped on the outer edge of the sole; and that the rupture of the internal lateral ligament or fracture of the internal malleolus was produced secondarily by the patient's attempts to walk; whereupon, the external malleolus being already broken, the foot was forced into valgus; but he stated that when fracture of the fibula followed a turning outward of the foot, the internal malleolus broke first and the fibula only secondarily. (Dupuytren: *Leçons orales de clinique chir.*, éd. 2^e, Paris, 1839, i, 327.)
23. Souligoux (*Bull. et mém. Soc. de chir. de Paris*, 1912, xxxviii, 1103) described two unpublished diagrams made by Tillaux to illustrate this "third fragment:" the anterior view showed the anterior tubercle of the tibia torn off, while the posterior view showed the posterior tubercle torn off. Apparently, then, it was Tillaux's opinion that either tubercle could represent his third fragment. A larger fragment from the inferior lateral margin of the tibia may exist, known to the French as the "intermediate fragment of Verneuil," but it is quite rare. This seems to correspond to what Roberts and Kelly (the advance proof-sheets of whose second edition, 1921, Dr. Roberts has very courteously sent me) describe as the "drunkard's fracture." Confusion is added to this matter by the statement of Thaon (*Bull. Soc. anat. de Paris*, 1870, xlv, 212), in presenting a necropsy specimen of a posterior marginal fracture, that he had often seen this posterior fragment in experiments made by Tillaux; and by the statements of Demoulin (*Bull. et mém. Soc. de chir. de Paris*, 1912, xxxviii, 1103), of Mauclairé (*Bull. et mém. Soc. de chir. de Paris*, 1912, xxxviii, 1141), and of Viallet (*Rev. de chir.*, 1912, xlvi, 690), that the third fragment of Tillaux may easily be mistaken in roentgenograms for a posterior marginal fragment. My own belief is that the third fragment of Tillaux is the anterior tibial tubercle and that the fragment composed of the posterior tubercle and that known as the posterior marginal fragment usually are indistinguishable.
24. Wagstaffe: *Saint Thomas's Hospital Reports*, London, 1875, vi, 43.
25. LeFort: *Bull. gén. de thérap.*, Paris, 1886, cx, 193.
26. LeRoy: *De la fracture marginale antérieure de la malleole externe*, Paris, 1887.
27. Bondet: *Thèse de Lyon*, 1899.
28. Quénu: *Rev. de chir.*, 1912, xlv, 1, 211, 416, 560.
29. Tanton: *Fractures du membre inférieur*, Paris, 1916.
30. Stimson: *Fractures and Dislocations*, 8th ed., Philadelphia, 1917, p. 450.
31. Murphy: *Surgical Clinics*, Philadelphia, 1914, iii, 2.
32. Murphy: *Surgical Clinics*, Philadelphia, 1916, v, 350.
33. Cotton: *Dislocations and Joint Fractures*, Philadelphia, 1910, p. 545.
34. Roberts and Kelly: *Treatise on Fractures*, Philadelphia, 1916, p. 580.
35. Rose and Carless: *Manual of Surgery*, 10th ed., London, 1920, p. 632.

36. Speed: Textbook on Fracture and Dislocations, Philadelphia, 1916, p. 772.

37. Nélaton: *Eléments de path. chir.*, Paris, 1844, vol. i, Preface.

38. From the Greek word for cheek, Quénu derives the adjective *génienne*, which he constantly employs in his classification and nomenclature of ankle fractures, inventing such terms as *bi-malleolaire géni-sus-génienne*, *géné-peronière* and *géné-supramalleolaires*.

39. Chaput (*Bull. et mém. Soc. de chir. de Paris*, 1912, xxxviii, 1192) says they are like Siamese twins.

40. The posterior tubercle of the astragalus may sometimes be fractured by direct violence as it is crushed against the posterior lip of the tibia in forced plantar flexion of the foot.

41. Humphry: *Treatise on the Human Skeleton*, Cambridge, 1858, p. 490.

42. Bland-Sutton: *Am. Jour. Med. Sci.*, 1888, xcv, 376.

43. Nancrede: *Philadelphia Med. Times*, 1880, x, 316.

44. Nancrede: *Maryland Med. Jour.*, 1880, vii, 76.

45. "Mixed" because involving the fibula both above and below the tibio-fibular joint.

46. Chaput (*Bull. et mém. Soc. de chir. de Paris*, 1906, xxxii, 1047) noted that among 130 cases of fracture at the ankle, of which he had studied the roentgenograms, 113 conformed to this oblique type of fracture of the fibula, against only 17 cases in which the fracture was clearly above the malleolus.

47. Lapointe (*Souligoux: Bull. et mém. Soc. de chir. de Paris*, 1914, xlv, 1042) quite justly contends that if fracture of the anterior tubercle can occur as an isolated lesion (as in the case he reported), it must be admitted it may also occur as the first lesion of a more complicated fracture.

48. Quénu: *Rev. de chir.*, 1907, xxxv, 897.

49. Long recognized as possibly due to pull of the biceps muscle. See a study by Lonhard (*Deutsch. mil.-ärztl. Ztschr.*, 1914, xliii, 219).

50. Huguier: *Union méd.*, Paris, 1848, ii, 120.

51. In addition to the 300 cases of fracture of the ankle listed at page 324, we have records of at least nine cases diagnosed as epiphyseal separations; but as in these the roentgenograms available showed no gross separation at the epiphyseal line of the tibia, we have not included them in our statistics.

52. Bonnet: *Traité des maladies des articulations*, Lyon, 1845, ii, 428.

53. Mention has already been made (p. 305) of isolated fracture of the external malleolus associated with fracture of the calcaneum (Fig. 23).

54. Once the lower end of the fibula is freed from the tibia, the interosseous membrane presents no obstacle to torsion.

55. Quénu: *Rev. de chir.*, 1912, xlv, 242.

56. Destot: *Lyon chir.*, 1912, viii, 245.

57. The criteria of diastasis from a roentgenographic study will be discussed by Dr. Bromer in his appendix to this paper.

58. Quénu: *Rev. de chir.*, 1907, xxxv, 897.

59. Millikin: *Ann. Surg.*, 1919, lxix, 650.

60. Gross (*System of Surgery*, 5th ed., Philadelphia, 1872, ii, 85) says Druitt refers to such a case, but I have been unable to find the original report to ascertain the exact lesions. Wendel (*Beitr. z. klin. Chir.*, 1898, xxi, 146) collected five cases of upward dislocation without fracture; but the exact lesions do not appear to have been determined definitely in any case.

61. Tanton (*Fractures du membre inférieur*, Paris, 1916, p. 165) refers to twenty cases recorded prior to 1916. As an isolated lesion it was first observed by Meissner (*Beitr. z. klin. chir.*, 1909, lxi, 136).

62. Earle: *Lancet*, 1828-1829, ii, 346.

63. Adams, in Todd: *Cyclopedia of Anatomy and Physiology*, London, 1835-1836, i, 161.

64. Malgaigne: *Gaz. méd. de Paris*, 1832, iii, 647.

65. Thaon: *Bull. Soc. anat. de Paris*, 1870, xlv, 212.

66. Nélaton: *Elémens de path. chir.*, 2d ed., Paris, 1847, iii, 296.
67. Andrews, in Ashhurst: *Internat. Encyclopedia of Surgery*, New York, 1883, iii, 707; also in *Internat. Clinics*, Philadelphia, 1897.
68. Chaput: *Bull. et mém. Soc. de chir. de Paris*, 1899, xxv, 776; *Les fractures malléolaires*, Paris, 1907.
69. Grashey: *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1907, xi, 152.
70. Plagemann: *Beitr. z. klin. Chir.*, 1911, lxxiii, 688.
71. Cotton, F. J.: *A New Type of Ankle Fracture*, *Jour. Am. Med. Assn.*, 1915, lxiv, 318.
72. In addition to the necropsy specimen which figures in Cooper's Plate XVII (reproduced here as Fig. 3), those described by Stimson and that of Thaon, already alluded to, there are in the Mütter Museum of the College of Physicians of Philadelphia three necropsy specimens showing this posterior marginal fragment. The most recent of these specimens has been in the museum for a period at least of forty years. (A description of these specimens will be published elsewhere by Dr. Bromer and myself.)
73. Lucas-Championnière: *Bull. Soc. anat. de Paris*, 1870, xlv, 212.
74. Rochet (*Rev. d'orthop.*, 1890, i, 269) produced it experimentally by dropping a weight of 60 kilograms, from a height on the upper end of the tibia while the foot was in plantar flexion.
75. Quénu: *Rev. de chir.*, 1912, vol. xlv.
76. Stimson: *Fractures and Dislocations*, 7th ed., Philadelphia, 1912, Plate 27.
77. Silhol: *Bull. et mém. Soc. de chir. de Paris*, 1916, xlii, 819.
78. Sear: *Med. Jour. Australia*, 1917, i, 526.
79. Gosselin: *Gaz. d. hôp.*, 1855, xxviii, 218.
80. Gosselin: *Bull. et mém. Soc. de chir. de Paris*, 1863, v, 147.
81. Malgaigne: *Fractures*, Paris, 1847, p. 818.
82. In addition to the references already given, the following, selected from more than 250 articles studied in the preparation of this paper, will be found of interest:
 - Bruns: *Die Lehre von den Knochenbrüchen*, Stuttgart, 1887, p. 57.
 - Farabeuf: *Précis de manuel opératoire*, Paris, 1909, p. 835.
 - Hamilton: *Practical Treatise on Fractures and Dislocations*, Philadelphia, 1860, pp. 443 and 685.
 - Messerer: *Ueber Elasticität und Festigkeit der Menschlichen Knochen*, Stuttgart, 1880, Plate 14, Figs. 3, 7 and 11.
 - Quénu: *Bull. et mém. Soc. de chir. de Paris*, 1906, xxxii, 943; *Rev. de chir.*, 1907, xxxvi, 62; *Bull. et mém. Soc. de chir. de Paris*, 1912, xxxviii, 1070; *ibid.*, 1919, xlv, 1142.
 - Scudder: *Treatment of Fractures*, 8th ed., Philadelphia, 1915, p. 545.
 - Tillaux: *Traité d'anatomie topographique*, 2d ed., Paris, 1878, p. 1023; *Gaz. d. hôp.*, 1886, lix, 89.

ORTHOPAEDIC SURGERY IN THE LAST SEVENTY-FIVE YEARS

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ORTHOPAEDIC SURGEON TO THE HOSPITAL

No branch of medicine was more forcibly impressed on the lay mind during the World War than orthopaedic surgery. Practically every wounded man at one time or another received the benefits of this speciality, and to many it was an introduction to a division of surgery that was regarded by them as a new form of treatment. In the following pages I shall, by giving a brief résumé of the history of orthopaedic surgery, show how this speciality has really grown hand in hand with the development of general surgery, always ready to pick up the subject where general surgery dropped it and eagerly studying those borderline diseases which general medicine turned over to general surgery, and in which general surgery has, in its turn, looked to orthopaedics for help.

While the events, from 1850, leading up to Lister's great discovery, in 1868, are the foundation of modern orthopaedics, as they are of modern general surgery, many men had devoted their lives to the study of the correction of deformities for years before Lister showed the possibilities of aseptic surgery. France is the true cradle of orthopaedic surgery, and Dr. Nicholas Andry, of Lyons, its founder and, in fact, the coiner of the word. His book, *Orthopædia* (ὀρθός, straight; παιδίου, child), was published in 1741, and is a most remarkable book on the prevention of deformities in children. He was an advocate of exercise for the prevention and cure of deformities and many of our modern orthopaedic practices can be traced directly to this pioneer.

A year after Andry's book was published, in 1742, Duhamel startled medical men by the results of his experiments on bone. An aristocrat, living quietly on his estates of Monceau,

Duhamel was of that rare type of mind which must submit any observation to the test of experiment. By a series of ingenious ones, extending over a period of years, he demonstrated satisfactorily to himself, at least, that periosteum was the mother tissue of bone. He was the man (and one not trained to medicine) who first demonstrated that bone grows in thickness by circumferential deposition of plate upon plate, very much as the trunk of a tree thickens with growth, and that a long bone grows in length at its extremities. The storm of adverse criticism that greeted Duhamel's discoveries was led by Albrecht von Haller, who believed that the arteries were the depositors and builders of bone, and who questioned, "How any anatomist could think that bone is formed only by periosteum." So the two great schools were established and the battle still rages merrily; the one holding that periosteum is a bone producer, and the other school maintaining that it is merely a limiting membrane, the skin of the sausage, as it were.

Jacques Delpech, of Montpellier, was the next important contributor to orthopaedic surgery. His great work, *l'Orthomorphie*, published in 1828-1829, proves him to have been the real founder of the theory that deformities are due to a lack of balance in the action of muscle groups. He was the first to make a deliberate attempt to apply gymnastics to the treatment of deformities of the human body. He mortgaged everything he owned to establish his Orthopaedic Institution, an establishment with a winter and summer gymnasium, baths, "flying bridge," "inclined plane" and a variety of other ingenious devices for active exercise in the correction of deformities. He did not confine his work to medical gymnastics, however, as, in 1816, he introduced to surgery a double innovation, the subcutaneous method of operation and the section of a tendon as a means of correcting deformity. Unfortunately, the results of the labors of Delpech were lost in the confusion following his assassination in 1832; we can only note that in the early decades of the Nineteenth Century one of the most famous surgeons of the day was convinced that the way to correct certain deformities was by active and persistent exercise.

Southern France was not alone in the advocacy of medical

gymnastics. Pehr Henrik Ling, the founder of the Swedish system of gymnastic exercises, had at the time of the death of Delpech, in 1832, succeeded, at last, in firmly establishing a Central Gymnastic Institution in Stockholm, where he dreamed that the youth of Sweden were to enter upon a new era of robust and happy health by having their muscles educated along with their brains. To him more than to anyone do we owe our present-day system of school gymnastics.

The question of "rest" or "motion" in the treatment of diseased or disabled bones or joints has always been a subject which has intrigued the orthopaedically-minded surgeon. In 1779 Jean Pierre David, professor of anatomy and surgery at Rouen, won the prize offered by the Academy of Surgery in Paris for an essay on this subject. "Time and rest will cure all joints," and "absolute rest is an imaginary thing," are two of his famous sayings. In spite of his advocacy of rest, he believed that there were conditions which called for motion, and he believed in active rather than passive motion. John Hunter, who began his active practice as an army surgeon some eighteen years before David's essay appeared, believed that the power to heal was inherent in the patient's tissue, but that the power to recover function was the property of the patient's will and brain. By a system of mechanical therapeutics he aided the return of function. He likened the surgeon to a gardener: each may prune and cultivate, splint and support; but each can only assist the power of growth, which is inherent in the tissue. John Hilton, another famous London surgeon, who lived in the first three-quarters of the Nineteenth Century, gave, as his contribution to the advance of orthopaedic surgery, his application of the principles of rest to the treatment of diseased and injured joints. He used the word "rest" in a wide sense—a scab which forms over an open wound, the exudates which appear on the surface of inflamed serous membranes, were Nature's means of protecting these parts from irritation, and thus providing them with rest. He designed many appliances for securing rest; probably his long hip splint being the best known today.

The question of subcutaneous tenotomies and subcutaneous operations in general was agitating the medical world in the

first half of the Nineteenth Century. We have seen that Delpech, in 1816, did the first known subcutaneous tenotomy of the tendo Achillis for club-foot; but it remained for Louis Stromeyer, in 1831, to really apply subcutaneous operations to the treatment of deformities. Among the hundreds of cases operated upon by Stromeyer was William John Little, who had suffered with an acquired club-foot from early childhood. So enthusiastic was Little over the success of this operation that he practically forced the idea down the throats of the skeptical London surgeons on his return to that city from Hanover. Little was the pioneer of orthopaedic surgery as a distinct speciality in England. In 1838 he founded in London the Orthopaedic Institution, which later became the Royal Orthopaedic Hospital. Because his attention was focused on muscles and tendons, he was able to recognize as a distinct condition that type of infantile spastic paralysis which bears his name. He continually emphasized the *credo* of the true orthopaedist, that the essential part of a cure is not the cutting, but the after-care of the case.

It was but a step for the surgical mind from the subcutaneous section of tendons to the subcutaneous section of bone. The first subcutaneous osteotomy was performed in Germany in 1852, but the honor of performing the first subcutaneous osteotomy in Britain fell to the son of William Little, Louis Stromeyer Little. A few years later, in 1871, William Adams performed the first subcutaneous osteotomy for ankylosis of the hip. The wave of enthusiasm for subcutaneous osteotomies had also swept over Scotland, and, in 1877, Sir William Macewen evolved the present method of supracondylar osteotomy for correction of knock-knee and bowleg.

“We cannot restore action to any machine, much less to that most complex of all machines, the human body, unless we know the arrangement and working of its parts.” The subject of innervation of muscles and the seat of reflex nerve action was a closed book until Marshall Hall’s epic, in 1833, on *Reflex Function of the Medulla Oblongata and Medulla Spinalis*, had established definitely the reflex action of the spinal cord. The healing of nerves after section—whether the cut ends took up the function of the nerve cell or acted merely as a

framework for new fiber growth—was explained in part in 1850, by that dark-skinned and smooth-tongued East Anglian, Sir James Paget. Augustus Waller, with his work on the divided nerves of the frog's tongue, further clarified the subject; but it remained for the experiments of Ross G. Harrison, of Johns Hopkins, in 1904, to definitely demonstrate that nerve fibers were the outgrowths from single nerve cells and not a combination of cells.

Duchenne, of Boulogne, was another famous contributor to the rapid development of orthopaedic surgery in the first half of the Nineteenth Century. He first conceived the idea of using the faradic current in the treatment of chronic joint and muscular conditions, and from this early work gradually grew to interest himself in determining the action of muscles by faradization. We are indebted to this remarkable observer for the first description, as a distinct disease, of "progressive muscular atrophy," "pseudohypertrophic paralysis" and "locomotor ataxia." A half-century later the work of Duchenne was further amplified by Dr. Charles Beevor, who, in 1903, published his method of studying both the normal and the disordered action of muscles.

Among the outstanding characters in orthopaedics in the latter half of the Nineteenth Century was Hugh Owen Thomas. Born of a long line of "bonesetters," he early realized the limitations of manipulations alone as a curative measure, and leaves us as his most important legacy the proper understanding of the word "rest." Immobilization is not rest. If the diseased part be compressed or the normal circulation be interfered with, the part is not at rest. Thomas was a mechanical genius and maintained a workshop in his home where he personally made and fitted his supports. In his splints he used the ensheathing principle, as crustacea are ensheathed with a bony framework which protects but does not compress the parts. He was sometimes bitter at the inability of his colleagues to grasp his ideas. He says: "Men admired my splints as if I were a blacksmith; but the principles on which they were framed they never could see." Thomas continually preached that rest must be "enforced, uninterrupted and prolonged." He believed that an overdose of rest was impossible.

Louis Bauer was the real founder of the school of orthopaedic surgery in America. A pupil of Stromeyer in Germany, he came to Brooklyn in 1852, later moving to St. Louis. Although his writings and work did not inaugurate a new movement, he brought us the best of European orthopaedic knowledge and firmly established the entity of orthopaedics in America. His contemporaries and rivals, Charles Fayette Taylor, Henry G. Davis and Lewis Sayre, all of New York, were greatly influenced by Thomas, as their well-known splints demonstrate. Taylor's spinal "assistant," first devised in 1873, is still the basis of most spinal braces, while the hip splint of Davis embodied all of the best ideas of Thomas. In 1874 Sayre applied the first plaster jacket for a tuberculous spine, and, although Thomas thoroughly disapproved of plaster casts, Sayre found that his jacket relieved the back pain and allowed his patient to walk about, as many other orthopaedists have since learned.

With the work of Duchenne and Beevor as a foundation, E. H. Bradford and R. W. Lovett, of Boston, have best applied modern physiology to the restoration of muscular function. Lovett's work on muscle reëducation, especially in cases of infantile paralysis, has become the accepted basis of treatment. The modern school of American orthopaedic surgeons seeks help from every line of new endeavor in the treatment of deformities. Compared to the European schools, we, perhaps, depend more on mechanical appliances than do our continental confrères; but on both sides of the Atlantic, orthopaedists are continually preaching that it is not alone the operation or the mechanical appliances that cures a deformity, but painstaking and meticulous after-care.

We have seen that the subcutaneous section of a tendon was done as early as 1816. With the advance of orthopaedic knowledge, surgeons realized that tenotomy did not always solve the problem of correcting a deformity. In 1881 Dr. Karl Nicoladoni, of Vienna, attempted a tendon transplantation. The result was unsuccessful and the operation was not tried again until 1892, when Dr. B. F. Parrish, of New York, performed an operation of a similar nature with success. The idea immediately became immensely popular. In 1897 Bradford reported 27 cases of transplantation, and in 1908 he

reported 500. Various ingenious modifications of tendon transplantations added to our ability to correct special deformities. In 1901 Dr. Tubby first transplanted the pronator radii teres from the flexor to the extensor surface of the forearm, thus changing it from a pronator to a supinator. In time the enthusiasm for tendon transplantation lessened. A muscle has its fibers arranged in its line of force. If the line of action is altered too radically by transplant, beyond the power of adaptation of the muscle, the muscle cannot contract and degeneration of its fibers sets in. Moreover, it is a most difficult feat to exactly equalize muscle pull in a transplantation; too much or too little is transplanted, and the pull of the transplanted muscle in its new position is not of the same amount as in its normal position; so that in many cases, after the lapse of a few years, either the old deformity recurs or a deformity in the opposite direction begins to appear, depending on the strength of the transplanted muscle and the strength of its antagonists. This fact has led orthopaedic surgeons to the development of the present method of correcting deformities by stabilizing the joints concerned. By an open or subcutaneous curettement of the cartilage from the joint surfaces, and a careful approximation of the bone ends with fixation for a sufficient time, a firm bony ankylosis results. This idea has been utilized in various parts of the body; in the foot by G. G. Davis and, later, by Hoke; in the spine by Hibbs.

All operations and appliances which aim to effect repair of the mechanical framework of the human body lie within the scope of orthopaedic surgery. The subject would, therefore, not be complete without at least mentioning the work done in the repair of fractures. As early as 1888, Lambotte, of Antwerp, was using plates which he screwed on to broken fragments of bone to hold them together. In 1893 Sir Arbuthnot Lane first used screws to hold the oblique fragments of a fractured tibia in apposition. Later he also designed and used plates for the same purpose. In 1911 Albee, of New York, first used living-bone grafts as internal splints. Ivory pegs, mule bone and screws of various kinds and materials have all been used, but the modern feeling tends to rest its hopes on the autogenous bone graft.

With our increasing knowledge of the human body, we have seen orthopaedic surgery develop in the last seventy-five years into a distinct branch of general surgery, specializing in the mechanical framework of the human body. It is an art devoting itself with infinite patience to the making of the "straight child;" trying to give back to the cripple that body "in the likeness of God," which its Maker intended as the rightful heritage of every human being.

A very brief summary of the orthopaedic work at the Episcopal Hospital for the year 1929 is appended.

In the orthopaedic dispensary 597 new cases were seen and 1960 old cases were treated, making a total of 2557 cases treated in the dispensary during the year; 123 appliances were secured through the social service department, amounting to an outlay of \$1650.65. A number of other braces, etc., were donated by various organizations and interested societies.

In the hospital 85 cases were taken care of in the beds at the disposal of the orthopaedic department.

46 operations were performed with no mortality, as follows:

Arthrodesis: shoulder-joint	1	Resection, old scar and plastic	
subastragalar	12	repair	1
midtarsal	1	Section: horizontal transverse	1
Amputation: mid-thigh	1	sternomastoid	1
toes	2	tendon pectoralis major	
Bone grafts: humerus	1	and subscapularis	1
(sliding) humerus	1	Sequestrotomy	2
Correction, hallux valgus	2	Stretching club-feet	5
Lengthening tendo Achillis	2	Suturing fractured patella	1
Excision, knee	2	Transplantation, tendon	1
Open reduction, congenital dis-			
location of hip	1		
Reconstruction, hip	6		
Removal, dislocated semilunar			
and fractured scaphoid from			
wrist	1		

Practically all operative cases had plaster casts applied, but excluding these, 80 plaster casts were applied during the year for immobilization in non-operative cases.

STOFFEL'S OPERATION FOR SPASTIC PARALYSIS, WITH REPORT OF THIRTY-TWO CASES.¹

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FORMERLY ORTHOPEDIC SURGEON TO THE HOSPITAL

IN an article on "The Surgery of Spastic Paralysis" (*Annals of Surgery*, May, 1918, lxvii, No. 5), I discussed briefly the surgical procedures employed in the treatment of spastic paralysis and made a preliminary report of cases operated on by the Stoffel method of peripheral neurectomy of motor nerves. The reader should study Adolf Stoffel's paper, "The Treatment of Spastic Contractures," which appeared in the *American Journal of Orthopedic Surgery*, May, 1913, x, No. 4. I performed my first Stoffel operation in February, 1914, at the suggestion of Dr. A. P. C. Ashhurst, whose assistant I then was at the Orthopedic and the Episcopal Hospitals. Since that time I have operated on 32 patients, in whom partial neurectomy has been performed 34 times on the popliteal nerve, 27 times on the obturator nerve, 6 times on the median nerve and 5 times on the sciatic nerve, a total of 72 operations. In this present paper I shall report these cases in detail and shall discuss the Stoffel operation and consider the position which it should take in the treatment of spastic paralysis.

It seems best to consider this subject in the manner in which the orthopedic surgeon confronts it in his daily experience. I shall not deal with the causes of spastic paralysis, nor with the various forms it assumes, except so far as they are related to the treatment.

CLINICAL VARIETIES. The patient presents evidences of spasticity of the muscles of one or more extremities. The muscle reflexes are exaggerated. The stronger groups of muscles usually have overcome by constant tension the

¹ Read at Toronto at the 1920 Meeting of the American Orthopedic Association. Reprinted from the *Journal of Orthopedic Surgery*, 1921, iii, 52.

feebler groups and have produced a certain state of deformity of the member. A condition of apparent contracture of the stronger muscles is present. In the upper extremity this is manifested in flexion of the elbow, the wrist and the fingers, and in pronation of the forearm. In the lower extremity it is manifested in the adduction of the thighs, the flexion of the knees and the plantar flexion of the feet. The amount of spasticity and the degree of contracture vary widely in different cases. In some, active motion of the affected member is entirely absent, *e. g.*, the foot is fixed in extreme plantar flexion, so that the patient is unable to move it. In others, the contracture is more moderate, and the patient has partial active motion, *e. g.*, he may be able to dorsiflex his foot to a right angle and plantarflex it to the normal limit. In still another group of cases active motion may be almost or altogether complete, but the patient has difficulty in originating and carrying out the motions, *e. g.*, the hand and fingers may be held usually in a position of flexion, but the patient is able, after some delay and possibly with the assistance of his other hand, to extend his wrist and fingers practically to the normal extent and actively to flex them again. In such cases no contracture is present, but the cerebral control of the member is lessened. Motions are delayed and slow and are not always to be depended upon when desired.

Many of the patients are seen by the surgeon before they have learned to walk. Others may be able to walk with considerable difficulty when assisted by a supporting hand. Others may walk fairly well, but usually with a certain degree of adductor, hamstring and Achillis contracture, so that the knees strike together or cross each other and are kept in partial flexion and the heels do not touch the ground. In a fairly large proportion of cases these patients are mentally deficient, and some are even completely idiotic. They are nearly always slow in learning to talk and to walk. Not infrequently, cases are met which have reached the age of puberty without being able to do either.

The tendency among orthopedic surgeons has frequently been to consider these backward children as more or less hopeless, and to decline to undertake treatment because of the

length of time and the amount of attention that are required, and because of the discouraging results that are often obtained; but certainly if no orthopedic measures are employed these needy cases will not improve of themselves. It is unquestionable that many cases can be very greatly helped, and that some improvement can be secured in almost all. In a low-grade idiot improvement may, indeed, be insufficient to justify a long course of treatment. But there are many cases on the borderline who should be given the advantage of reasonable doubt. If it is possible for the surgeon to enable these individuals to walk, a great deal will have been accomplished in adding to their usefulness and to the comfort of their families.

PRELIMINARY TREATMENT. The object of all treatment is to secure restoration or improvement of function. If a patient is unable to walk, the aim of the surgeon is to enable him to do so, even by the use of crutches and braces if necessary. If he is able to walk with difficulty, the object is to improve his locomotion. If the disability is in the upper extremity, treatment is for the purpose primarily of securing better function of the hand, so that the patient may have an enlarged field of usefulness in life and, secondarily, of improving the appearance of the hand and of relieving painful or uncomfortable spasm of the muscles.

In examining these cases, the surgeon should note the extent of the spastic paralysis, the comparative difference in the strength of antagonistic muscles or groups of muscles, the degree of contracture and its condition, as to whether it is permanent or temporary, the amount of disability in the member, the mentality of the patient and his social condition. One should know also whether or not the patient has been neglected, or whether he has received intelligent treatment. The surgeon may then outline the course of treatment which may be necessary to arrive at the definite end of return or improvement of function.

If the patient can remain under the care of the surgeon as long as is desired, a preliminary course of treatment should be undertaken before any operative procedure is considered. If the circumstances are such that whatever can be done should

be done quickly, then immediate operation may be performed. If a patient has already had careful treatment, which has been largely in the way of prophylaxis to prevent deformity, the preliminary treatment may be dispensed with. This treatment is undertaken for the purpose of improving function as far as possible before surgical operation is resorted to, and to enable the surgeon the better to decide what operation, if any, may be required. If the patient has a marked talipes equinus, so that he stands or walks upon his toes, and has no active dorsiflexion of the foot, the surgeon may be unable at once to decide what operation may be necessary, or how far the operation should go, in order to secure a good result. The anterior muscles of the leg appear to be completely paralyzed; but if, with or without an anesthetic, the foot is dorsiflexed and the knee extended and the extremity put in plaster of Paris in an overcorrected position for a period of four to six weeks, it may be found at the end of this time that the anterior leg muscles have recovered considerable power, and that the patient has considerable active dorsal and plantar flexion of the foot. If this improvement is maintained by means of a brace, and if massage and painstaking active exercises are instituted, with possibly the use of the galvanic current, still further improvement may be secured. It may be found that no operation is necessary. If, however, the foot shows marked tendency to return to its former position of equinus, or if sufficient improvement has not taken place, the surgeon is then in a position to note to what extent the Stoffel operation should be carried. The same observation holds true for deformity of the upper extremity.

It must be borne in mind that in spastic paralysis the element of spasticity far overshadows the element of paralysis. Indeed, a real or complete paralysis is very rarely present. All muscles are spastic, but the strong muscles have overcome their weaker antagonists and produced a deformity. The weaker muscles are thus still further weakened by overstretching and by being placed in a bad mechanical position, and the stronger muscles become still more spastic by the approximation of their points of origin and insertion. One object of all methods of treatment, including the Stoffel

operation, is simply to secure more or less muscle balance, so that the weaker muscles or muscle groups may not be overcome by more powerful antagonists, but may be able to exercise their proper function. In normal individuals the play between antagonistic groups of muscles is under cerebral control, but in cases of spastic paralysis this control has been more or less lost. When the muscle balance has been approximately restored, the patient may regain almost normal active motion of the member or he may secure but feeble and delayed motion. In still other cases the amount of active motion secured may be very slight, but the patient is benefited by the removal of the deformity and the consequent improvement of function. In addition, the cosmetic appearance has been greatly improved, a consideration of no mean value in the upper extremity.

If the surgeon, by means of the Stoffel operation, has secured more or less muscle equilibrium, he must not consider his task ended, for a long and careful course of after-treatment is necessary. The weaker muscles, which have so long been at a disadvantage, must be carefully strengthened and trained. For this purpose, massage, galvanic electric current, manipulation of the joints and, above all, careful active exercises must be carried out. Braces may be necessary to prevent relapse. It may be found that too little resection of the nerves has been done and a second operation will be necessary. Of course, when the mentality of the patient is fair or good, the improvement will be much more rapid. Idiotic children may be unable to assist in any way and the surgeon may have to be content with the elimination of the deformities present. Such patients may possibly learn to walk with the use of braces and by the employment of an apparatus such as a wheel-crutch.

PURPOSE OF THE STOFFEL OPERATION. We consider now the operation in itself. As has been stated, the deformity in the patient is due to muscle unbalance. The disability is due in part to the deformity and in part to the spasticity and greater or less interference with normal cerebral control of the muscles. The operation aims to remove the deformity and as well to break the vicious circle of the peripheral arc, which

produces the spasticity. In my article on spastic paralysis, previously referred to, I have described the establishment of this vicious circle and the means employed to break it. I shall not discuss it here; it is sufficient to say that the Stoffel operation does more or less cut this circle and relieve to a greater or less extent the spasticity. When this has been accomplished it is found that the brain in most cases is able to exercise more or less control over the peripheral arc. Therefore, we may say that the object of the Stoffel operation is twofold: to remove deformity, *i. e.*, contracture, and to secure better cerebral control of the members. The operation consists in the excision of a portion of the nerve supply to the strong and contracted muscles. This produces a partial or complete paralysis of these muscles. The amount of paralysis or weakening of a muscle group should be sufficient to cause a muscle balance between this group and the antagonistic muscles. If this be overdone, the antagonistic muscles may in turn overcome those which were formerly the stronger and produce a deformity opposite to the original one. Therefore, the chief element in the operation is the matter of judgment as to how large a portion of the nerve supply to any muscle or any group of muscles as a whole should be excised. This is somewhat a matter of experience, and until this experience has been gained the surgeon should err by doing too little rather than by doing too much, as he can always return to resect more nerves if necessary, but he cannot replace nerves already resected. Let me here emphasize, again, the importance of preliminary treatment to enable the surgeon to judge how far he should proceed with his nerve resection.

TECHNIC OF THE OPERATION. Let us now consider the method of operation upon the various nerves.

First, the Median Nerve. The common deformity in the upper extremity has been already described. I have not found it necessary to operate for flexion contracture at the elbow. But it is possible in certain cases that a portion of the nerve supply to the biceps muscle might require resection. The median nerve is exposed in the bend of the elbow. The incision need not be more than 2 inches in length. The nerve is freed from surrounding tissue, and partially retracted out

of the wound, and supported on a grooved director or a hemostat, placed transversely. On the anterior aspect of the median nerve, adjacent to the biceps muscle, is to be seen a nerve tract or cord. A portion of this separates itself from the median nerve and goes directly to the superficial head of the pronator radii teres. This is the first branch to leave the median nerve in the flexure of the elbow. This branch should be entirely resected for a distance of several inches. It can be cut off below near the point where it enters the muscle, and can be dissected up from the median nerve any desired distance. The remainder of the tract already described consists of the nerves to the flexor carpi radialis, the palmaris longus and the deep head of the pronator radii teres. These bundles of nerves are to be freed from the remainder of the median nerve and from one another and to be tested by the electrode. In severe cases of pronation and flexion contracture all of these nerves should be excised. In less severe contracture one may split the nerve to each muscle and may resect approximately one-half to three-fourths of it. The nerve to the pronator quadratus is to be found in the dorsal portion of the median nerve. Usually it should be left intact, but in very severe cases it may be resected. In none of my cases have I found this resection necessary. The median nerve is now further separated into its constituent bundles. It will be found that the nerve supply to the flexors of the fingers is on the dorsal and ulnar aspect. These bundles should be freed from the remainder of the median nerve, separated from each other and tested by the electric current. Such portion of them may be removed as is demanded by the severity of the contracture. The remainder of the median nerve consists of the sensory tracts and the motor nerves for certain intrinsic muscles of the hand. It is thus found that the median nerve consists of a definite number of nerve bundles or funiculi, which, like cables, are bound together. The tracts to the various muscles always occupy the same relative position in the nerve; in other words, the nerve has a definite topography or internal structure which is always the same. The surgeon will soon learn by observation the position of these various bundles, and may in time learn to dispense with the use of the

electrode, but in his earlier cases it is advised that he test always each bundle after it has been isolated to determine which muscle it supplies. One may use the ordinary brain electrode, or one may have made a special electrode for this purpose. It will be found that very slight stimulation is necessary to secure response in the muscle. If a strong current is used, there is danger of its spreading from one bundle to another, thereby confusing the surgeon as to which bundle of nerves he has isolated. The least strength of current necessary to produce muscular contraction should be employed. It is found that these spastic muscles respond to slighter stimulation than do normal ones. The surgeon has now produced a certain amount of paralysis of the muscles of pronation and of the flexors of the wrist and fingers. He has left, of course, untouched the ulnar nerve, which also supplies a portion of the flexors of the wrist and fingers. It may be attacked in a similar manner at a later operation if necessary.

Second, the Obturator Nerve. The anterior branch of the obturator supplies the gracilis, the adductor longus and all or a portion of the adductor brevis. The posterior branch supplies the obturator externus, the adductor magnus and, at times, the adductor brevis. Certain other muscles aid in the adduction of the thigh, notably the pectineus, which is supplied by a branch from the anterior crural, and the hamstring muscles which are supplied by branches of the sciatic. The dorsal portion of the adductor magnus frequently receives nerve supply through the great sciatic nerve. The adductor magnus is frequently divided into two distinct portions. The upper part of the anterior portion is usually quite separated from the rest of the muscle and has been termed the adductor minimus. It is, therefore, clear that if the anterior branch of the obturator nerve be excised, a considerable degree of adduction remains in the muscles supplied by the posterior branch and by the other nerves just mentioned. If the posterior branch be excised in addition to the anterior, the patient still possesses power of adduction through the pectineus, the dorsal portion of the adductor magnus and through the hamstrings in certain positions of the thigh. Therefore, in moderate degrees of spasticity the entire anterior branch of the

obturator nerve is excised; in severe degrees both branches are excised. I have found it necessary only on one occasion to excise the posterior branch as well as the anterior.

The technic of the operation is as follows: An incision, 2 or 3 inches in length, is made from the pubic spine downward along the tendon of the adductor longus. This tendon is identified and a blunt dissection is made by the handle of the scalpel or with the fingers along its inner margin. Dissection should never be made through muscle substance, but always in the cleavage planes between the muscles. The tendon of the adductor longus is then retracted outward and the anterior branch of the obturator nerve is clearly seen running in the intermuscular fascia. If one division only of the anterior branch is observed, it may be lifted upon a small hook and the blunt dissection carried proximally until the main stem of the nerve is isolated. The main stem is found to divide usually into three branches. The dry dissection is carried up to the obturator foramen. The posterior branch of the obturator can then be seen issuing from the foramen or running backward behind the anterior fibers of the obturator externus and behind the adductor brevis. If the anterior branch alone is to be resected, the main stem is clamped by a hemostat and is divided above the hemostat. The various branches are then divided several inches lower, as they are seen entering their respective muscles. If the posterior branch is to be resected, also, it is removed in a similar manner. One or two sutures are placed in the deep fascia and the skin wound is closed.

Third, the Sciatic Nerve. This is attacked to overcome contracture of the hamstring muscles. The nerve is exposed without difficulty by an incision begun at the gluteal fold and running downward for 4 or 5 inches and placed about midway between the tuberosity of the ischium and the great trochanter. After the deep fascia has been incised, the long head of the biceps muscle is identified. This muscle is then retracted inward and the dissection continued along its edge until the sciatic is encountered. On the median aspect of the nerve is found the cord which supplies the long head of the biceps, the semimembranosus and the semitendinosus. This tract or

cord is separated from the main trunk of the sciatic nerve, is lifted by a hook or an elevator, and is then dissected into its component parts. These three nerves are identified by means of the electric needle. In moderate degrees of spastic contracture the nerve to the biceps and the nerve to the semimembranosus are excised completely. In severe cases of contracture the nerve to the semitendinosus is split and about one-third of it is resected. The surgeon, therefore, in the first instance, leaves intact the short head of the biceps and all of the semitendinosus, and in the latter instance the short head of the biceps and on the median side the greater part of the semitendinosus. These two muscles are sufficient to secure active flexion of the knee, but their united power is not sufficient to overcome the extensor muscles of the thigh.

Fourth, the Internal Popliteal. Operation is made to correct pes equinus or pes equinovarus. This nerve is exposed in the center of the popliteal space. Here it lies only superficially and is easily approached. Bloodvessels and other structures lie deeper. The nerve is lifted from the wound and placed across a grooved director. It is well freed upward and downward from surrounding tissue. The first branch seen to be coming from the nerve leaves on the internal and posterior aspect and immediately pursues a superficial course. This is the sensory branch, called the nervus cutaneus suræ medialis or the tibialis communicans. The next two nerves to leave the main trunk are the nerves to the outer and inner heads of the gastrocnemius muscle. Running beside these two nerves is the nerve to the dorsal portion of the soleus, and the nerve to the plantaris. The common tract containing these nerves lies on the dorsal or superficial aspect of the internal popliteal nerve. The ventral portion of the soleus muscle is supplied by a special tract which lies on the antero-external aspect of the nerve. The nerve to the tibialis posticus will be found on the posterior or the postero-external aspect. That for the flexor longus digitorum on the posteromedian aspect. In moderate cases of pes equinus the two nerves to the heads of the gastrocnemius along are resected. In slightly more severe cases a portion (approximately one-half) of the nerve tract to the dorsal portion of the soleus is also excised. In still more severe

cases this entire tract is excised. If it is found in the severe cases that the flexor longus digitorum is materially aiding in producing the deformity, a portion of its supply must also be resected. If the tendency to pes varus is marked, a portion of the supply to the tibialis posticus must be taken. After the nerves have been resected, the remainder of the internal popliteal nerve is dropped back into its position, where it lies embedded in fat. The deep fascia is brought together by a few sutures and the skin wound is closed.

When the patient is under the full influence of the anesthetic it is usually found that all contractures which were present before operation have disappeared. In other words, the contractures were spastic contractures, and not atrophic, such as occur in infantile paralysis. This demonstrates the impropriety of the old operation of tenotomy or tendon lengthening, whereby tendons of muscles were lengthened where no actual shortening was present and an opposite deformity was produced in many cases, *e. g.*, a pes equinus was transformed into a pes calcaneus, which is the more disabling of the two. If it is found at the time of operation that contracture of the tendons still is present, then we know that real shortening has occurred, and tendon lengthening should be done in addition to the nerve resection. However, this tendon lengthening should be performed only in case the surgeon is unable by manipulation to stretch the tendon. I have not infrequently found contracture remaining during the anesthesia, but I have almost invariably been able to overcome it by forcible correction.

AFTER-TREATMENT. My method of after-treatment varies somewhat from that recommended by Stoffel. He employs starch bandages, splints and sandbags in all cases to maintain overcorrection, and avoids the use of plaster of Paris. In all cases of spastic paralysis of the lower extremities in which the contractures disappear spontaneously under the influence of the anesthetic, I employ no after-fixation. If forcible correction is necessary to reduce the contracture, then I fix the member in an overcorrected position in a light plaster cast for two or three weeks. If I find within a week after operation that some tendency to contracture persists in the

former class of cases, I may then put the member in a plaster cast, with or without the use of an anesthetic, for a short time. It is often remarkable and astonishing, on observing a patient the day following the operation, to find how completely his deformities have disappeared, and how considerable a degree of active motion he has in the direction where formerly he had little or none. If the proper preliminary treatment has been employed before operation, the less likely is one to require any means of fixation after operation. As soon as the wounds have healed, which is at the end of a week, the patient is got out of bed and is sent to the gymnasium, where he receives careful instruction in muscle-training and in walking. The child is hereafter encouraged to walk as much as possible without becoming fatigued. He is no longer a bed-patient, unless walking is absolutely impossible for the time being.

During my early operations I handled the nerves with great gentleness, making dissection of the component parts with extreme care. I soon found that this is unnecessary. While the nerve should not be handled roughly, one need not delay the operation by extremely delicate dissection. Occasionally, if the surgeon abuses the sensory tracts in the nerve, the patient may complain for some days after operation of some numbness or tingling in the course of the distribution of these tracts. This is more apt to be the case in the operation upon the median nerve. I have never observed it in operating on the internal popliteal nerve, and but once have I seen it in operation on the median, when a complete dissection was made of all the tracts of the nerve. Ordinarily, one does not touch the sensory tracts. He is familiar enough with the topography of the nerve to isolate the tracts desired with but little dissection of the remaining portion of the nerve.

The after-treatment has been mentioned. The patient may require no apparatus. If any measures are needed to correct a pes valgus, or a returning inclination to pes equinus, appropriate apparatus or pads in the shoes should be prescribed. In treating the upper extremity, I think, greater care must be exercised. It will usually be found better to apply a light cast immediately after the operation, with the forearm and the hand in a position of overcorrection. Afterward the wrist and

fingers should be kept on a splint in a hyperextended position for a period of possibly three or four months. The hand should be taken from the splint frequently during the day for treatment and exercise. A little later, the splint may be cut off so that free motion of the fingers is permitted, while the wrist is still maintained in hyperextension. Care must be taken always to avoid overfatigue of any of these patients. While they are encouraged to make active movement, yet it must not be overdone. This is a very important note of warning.

INDICATIONS FOR OPERATION. It is clear from what has been said that not every case of spastic paralysis is suitable for operation. Those are the most suitable which have a fixed deformity due to contracture of one or more of the muscle groups which have been enumerated in the various operations described above, whether they be due to a cerebral or to a spinal lesion. These cases of spasticity whose members are not in any fixed position, but assume first a position of flexion and then one of extension, or those patients with athetosis, are not suitable for operation. However, there is one group which may be called that of mild contracture. In this a certain mild deformity is usually present, but it can be voluntarily corrected or can be readily corrected by manipulation. When the patient becomes excited, the spasm and the deformity are exaggerated. This class is suitable for the Stoffel operation, but only a small portion of the nerve supply to the stronger muscle groups should be excised. The operation is contraindicated, furthermore, in the most severe phases of Little's disease, with diffused spasm in almost the entire body, and in progressive diseases, such as progressive multiple sclerosis, and those extreme cases of idiocy in which the patient would never be able to walk even if the deformities were corrected and the spasticity reduced. Hemiplegias, whether in children or in adults, may often be found suitable for operation.

REPORT OF CASES

No. I. R. J., aged seven years. Spastic paraplegia, present since birth.

February 6, 1914. Walks on toes, crosses legs in walking and is very unstable. Operation: Tenotomies of adductors, hamstrings

tendo Achillis of left leg, and Stoffel's operation on anterior branch of the obturator and portion of the branches of the sciatic to the hamstrings and portion of branch of the internal popliteal to gastrocnemius and soleus of right leg.

January 9, 1915. Very much improved. Walks fairly well with braces, heels come to the ground. Knees are not flexed; toes no longer dragged.

September 23, 1916. Braces removed.

August 25, 1917. Right foot is good, toes straight, but there is slight tendency to equinus. Left foot is in marked calcaneocavovalgus. Brace reapplied to left leg to hold foot straight.

March 9, 1918. Right foot is firm on the ground, with knees straight. The thighs are well apart. Good active flexion and extension of ankle. On the left side there is no adduction and no contracture at the knee, but the foot is in marked calcaneovalgus.

June 28, 1918. Operation: Left foot, horizontal transverse section and shortening of tendo Achillis.

December 13, 1919. Left foot is solid. No lateral deformity. The heel comes to floor at the same time as the ball of the foot, with the foot in slight dorsiflexion. Has a few degrees of active motion in the ankle. Knee is straight. Legs are well apart in walking. No tightness of adductor tendons on either side. In the right foot there is active dorsiflexion to about 90 degrees. The entire foot rests on the floor when standing with knee extended, but when walking the toes strike the ground first and then the heel comes to the ground. She walks fairly well without any support.

RESULT. Walking very considerably improved. The tenotomy of the left tendo Achillis led to a marked pes calcaneovalgus, which required a later operation to correct.

No. 2. E. W., aged five years. Spastic hemiplegia, which followed convulsions when eight months of age.

January 26, 1914. Marked disability of right arm and right leg.

March 25, 1914. Operation: Tenotomy of tendo Achillis.

July 27, 1914. Walks well, heel comes to the ground. The right hand and fingers are moderately flexed and the thumb is adducted and flexed.

September 20, 1914. Operation on median nerve.

October 26, 1914. Active motion of elbow 55 to 140 degrees. Active extension of wrist to 180 degrees. Active extension of fingers to normal. Thumb remains flexed into the palm. Inability to flex fingers completely.

July 17, 1916. Active motion of elbow 45 to 180 degrees. Hand is fairly straight. Thumb is flexed into the palm. Inability to make complete fist. Passive supination normal, active supination about one-half normal. Walks with heel on ground and foot straight.

February 14, 1918. Foot in good position, heel on ground, walks well. Passive supination complete; active, about one-half normal. Some tendency for hand to be flexed at wrist. Passive extension of fingers, wrist and thumb normal. Active extension partial. Brace advised to maintain hand in hyperextension.

January 20, 1919. Grasp is good. Wrist, fingers and thumb are held slightly flexed, and the thumb is adducted into the palm. At times she is able to extend the fingers to a straight line and the wrist almost to a straight line. Passive hyperextension of fingers. Wrist and thumb is normal. Patient has benefited by the operation, but has lacked development of the extensor muscles by exercises.

RESULT. Tenotomy of tendo Achillis satisfactory. Function of the hand considerably improved; appearance of hand very greatly improved.

No. 3. F. E., aged thirteen years. Paraplegia. June 26, 1914. Was operated upon nine years ago by Dr. Williard, who did tenotomies at the hips and tenotomies of hamstrings and probably of the adductors. Able to stand with support to balance him. Can walk sideways when supported by both hands. Stands with marked back knee, pronated feet, legs far apart. Right upper extremity fairly normal; left upper extremity—cannot raise arm above head or to behind back. Triceps and biceps weak. Grasp is weak. Wrist slightly flexed. Active extension of wrist limited to 180 degrees. Thumb cannot be abducted. Distal phalanges are in hyperextension, lead-pipe spasticity of elbow. Lower extremity: Slight motion, active at hips. No active flexion of knee. Slight active extension. No motion of ankles. Tendo Achillis contracted. Marked ankle clonus, marked quadriceps clonus.

July 4, 1914. Stoffel operation on both internal popliteals. Right leg: A portion of the nerve to the soleus, all of the nerve to one head of gastrocnemius, two-thirds of nerve to other head of gastrocnemius excised. Left leg: Both nerves to the gastrocnemius and two-thirds of nerve to soleus excised.

July 15, 1914. Active dorsiflexion of feet. Can walk with little support.

August 8, 1914. Discharged. Fairly good use of legs.

RESULT. Improved. Deformity removed. Good active dorsiflexion of feet secured.

No. 4. T. F. *Spastic hemiplegia since birth. July 17, 1916. Operation: Median nerve. Patient left the hospital at the end of several months considerably improved. He has not been heard from since.*

RESULT. Considerably improved. Have been unable to follow up the case.

No. 5. W. G., aged twelve years. *Spastic hemiplegia, left side; occurred when two and a half years of age.*

July 13, 1914. Raises arm only partially above head and places it behind him with difficulty. Shoulder elevated. Trapezius slightly contracted. Biceps and triceps have fair power. Arm is pronated, and cannot be supinated beyond the midposition. Wrist is flexed. Thumb and fingers are flexed into the palm. No active extension of wrist. No motion in fingers, except for a little additional adduction and flexion. Walks on the toes of his left foot, tendo Achillis is contracted. There is no active motion in the ankle clonus present. Has hollow foot and hyperextension of great toe.

November 4, 1914. Operation: Left median nerve and the internal popliteal. It was found at the time of operation that the tendo Achillis remained in partial contracture. It was lengthened and the extensor proprius hallicus was transplanted to the head of the first metatarsal.

December 7, 1914. Hand and fingers are straight. The thumb is not flexed into the palm. Arm can be supinated only to midposition. Brace applied to maintain extension of wrist.

September 20, 1915. Uses hand quite well. Picks things up. Has a good grasp. Carries his schoolbag in his hand and holds things well. Active extension of fingers almost complete. Can make a good fist. Has slight control of thumb, but it usually remains adducted into the palm. No active supination. Can extend wrist feebly when elbow is extended. Slight radial deviation of the hand. Foot in good position. Patient has been greatly benefited by the operation. Formerly he had not the slightest use of his hand; now he uses it well for many purposes. Walking has been improved very much.

RESULT. Very marked improvement in function of hand. Marked improvement in walking.

No. 6. W. A., aged twenty-seven years. *Spastic paralysis of right lower extremity, probably a lateral sclerosis.*

July 13, 1914. Walks with very spastic gait, with knees striking together. Uses cane.

November 14, 1914. Operation: Stoffel's, on anterior branch of right obturator nerve.

December 14, 1914. Walks better. Is less spastic, can actively abduct thigh a little. Has foot-drop.

February 8, 1915. Spasticity has increased. Patellar reflex and ankle clonus marked. Contracture of tendo Achillis. No active dorsiflexion of foot. Unable to lift foot from ground in walking. Weakness of flexors of thigh.

February 11, 1918. Patient has been working for some time. Walks fairly well with cane. Knee-jerk is exaggerated. Can lift foot from ground in walking. Slight active and passive abduction of thigh. Good flexion of hip. Full active extension of knee.

January 26, 1920. Patient reports by letter that he is working in New York City and that he is very greatly improved.

April 12, 1920. Patient has been working as a shoemaker and has been making \$26.00 a week. He walks quite well with the use of a cane and with his legs well apart. No tendency to adduction deformity, otherwise condition remains about the same.

RESULT. Operation on the obturator nerve has proved satisfactory.

No. 7. F. W., aged fourteen years. *Spastic paraplegia. Fell down stairs when ten years of age, was unconscious for a time, then had diphtheria; lameness was noted three weeks later.*

January 4, 1915. Typical spastic gait. Walking on toes, crossing knees and not lifting feet from the ground.

January 27, 1915. Operation on both anterior obturators.

March 8, 1915. Patient walks much better, with knees apart, feet flat on the ground. Feet do not drag in walking.

Patient died before reporting again at clinic.

RESULT. Improved by the operation.

No. 8. C. J., aged twenty-three years. *Spastic hemiplegia; resulted from bullet wound of head ten years ago.*

March 20, 1915. Contracture of tendo Achillis, pes valgus on standing, marked ankle- and knee-clonus.

March 24, 1915. Operation: Stoffel's, on internal popliteal.

May 1, 1915. Patient states that he is much improved. Walks very much better. In good position. Is exercising the anterior leg

muscles, which are getting stronger. Active dorsiflexion of foot is good.

February 26, 1916. Good dorsiflexion of foot. Heel comes to ground and foot is straight. He has had excision of keloid scar in the popliteal space resulting from the old excision.

Patient died January 16, 1918.

RESULT. Greatly improved by operation.

No. 9. F. Y., aged fifteen years. Spastic hemiplegia, left side. Followed diphtheria when six years of age. Stoffel's operation by Dr. A. P. C. Ashhurst, June 13, 1914: partial excision of both nerves to the gastrocnemius and one to the soleus; excision of nerve to the pronator radii teres and partial excision nerve to flexors of hand and fingers.

February 8, 1915. Heel comes to floor in walking, but there is moderate pes valgus. Hand is still flexed and the arm is pronated, but is not so spastic as before operation.

April 12, 1915. Walks fairly well. Hand less spastic, but remains in flexed position most of the time.

April 21, 1915. Operation: Stoffel's, on median nerve.

July 12, 1915. Voluntary partial extension of wrist and fingers, and partial active pronation and supination. Partial voluntary flexion of fingers; does not use her hand for anything. Patient cannot be traced. Final result not known. Immediate result was unsatisfactory as to function of hand, but it relieved the marked spasticity of the flexors of the fingers and the wrist.

RESULT. Moderate improvement.

No. 10. M. O'D., aged six years. Spastic hemiplegia; probably followed whooping-cough and convulsions when two years of age.

May 11, 1915. Walks with great difficulty. Contracture of tendo Achillis and foot-drop and pes varus. Toes alone come to the ground.

June 28, 1915. Operation: Stoffel's, on popliteal nerve. Excision of branches to the two heads of gastrocnemius; excision of portion of nerve to tibialis posticus.

July 6, 1915. Passive dorsiflexion well beyond 90 degrees. Heel on ground in standing and walking. Active flexion of some of the toes, which was lacking before. Foot is held almost at a right angle when the stocking is put on. No varus.

February 26, 1918. When standing, foot is on floor and in good position; when walking there is a slight tendency to walk on toes. Passive dorsiflexion to 80 degrees. Active dorsiflexion incomplete.

Mother states that child walks much better and is a great deal better in every way.

January 12, 1920. Mother states that she has steadily continued to improve. She walks quite well. Foot comes squarely to the floor and heel touches the ground. No lateral deformity. Fair active flexion and extension of ankle. Knee is held slightly flexed in walking, but patient is able to extend it fully. No ankle clonus. No varus.

RESULT. Greatly improved. Whereas formerly she was scarcely able to walk she now walks quite well.

No. 11. E. M., aged fourteen years. Spastic hemiplegia. (Right side has been paralyzed since birth.)

June 14, 1915. Cannot raise arm fully above head. Fair power in deltoid and trapezius. Contracture of both axillary folds. Limitation of external rotation. Biceps and triceps have fair power. Wrist is held in extreme flexion of 90 degrees. No active extension; passive extension greatly limited. Arm is pronated and the fingers and thumb are flexed into the palm. Does not use her arm for any purposes. Marked equinovarus of the right foot.

July 21, 1915. Operation: Stoffel's, on the right popliteal nerve and lengthening of the flexor tendon at the wrist.

October 25, 1915. Foot is in good position. Walking is much improved. Feeble active extension of wrist and fingers.

December 6, 1915. Uses right hand to dry dishes; can grasp objects feebly with thumb and fingers. Wrist and fingers remain fairly well extended. Slight active extension of fingers.

February 18, 1918. Walks quite well; toes strike ground, but heel touches when bearing weight. Wrist and fingers are flexed and cannot be fully straightened. Active extension of fingers when wrist is flexed.

February 27, 1918. Operation: Stoffel's, on median nerve.

March 18, 1918. Passive hyperextension of fingers and wrist is normal. Slight active extension.

April 18, 1918. Wrist and fingers are straight. Has active extension of fingers but none of the wrist.

October 28, 1918. Hand and fingers remain extended. Normal passive hyperextension. Slight active flexion of wrist. No active flexion of fingers. Good adduction of thumb, but no opposition. Walks with but slight limp with heel on the floor and no deformity of foot.

March 31, 1919. Active extension of wrist almost to a straight line. Active extension of fingers not quite complete at M. C. P.

joints. Good active flexion of wrist, but slight active flexion of fingers. Walks well, with heel on ground.

RESULT. Walking much improved. Function of hand moderately improved. Appearance of hand very greatly improved.

No. 12. C. C., aged nine years. Spastic paraplegia since birth. Did not walk until six years of age.

September 12, 1914. Walks with great difficulty; walks on toes and crosses legs.

September 26, 1914. Operation by Dr. Ashhurst: Excision of portion of anterior obturators and portion of internal popliteals to the gastrocnemius and soleus.

April 25, 1915. Walks very much better; heels come to the floor but the toes strike the ground first. Knees are flexed and thighs still slightly adducted.

September 26, 1915. Operation: Stoffel's; complete resection of nerves to both gastrocnemii and remainder of anterior obturators.

June 3, 1916. Much better, but still marked spasticity of adductors and hamstrings. Heels come to the ground. Walks much better. Father states that he is improved 50 per cent.

August 16, 1919. Walks with knees slightly apart and feet flat on the floor. Has tendency to double pes valgus. He continues steadily to improve and is walking better all the time. Adductors are well relaxed. Knees are straight. Good dorsiflexion of feet.

RESULT. Marked improvement in walking.

No. 13. C. W., aged twelve years. Spastic diplegia since birth. Was born at seven months.

November 27, 1917. Spastic paralysis of all four extremities. Has never walked. Knees cannot be fully extended. Passive flexion of feet to right angles. Unable to walk even with crutches. Stands on toes; adductors are spastic; knees are held tightly together and can scarcely be separated. Mental condition backward.

February 6, 1917. Operation: Stoffel's; excision of nerves to both heads of gastrocnemii and portion of sciatic supply to solei.

April 12, 1917. Operation: Stoffel's; excision of both anterior obturators and of portion of sciatic supply to the hamstrings.

March 9, 1918. Remained in hospital for five months after operation, taking exercises. He now walks with crutches, one foot forward at a time. Feet are flat on the floor; slight tendency to valgus. Knees are held slightly flexed; active flexion of feet to beyond right angle. Extension to about 135 degrees. Knees are

well apart in walking, active and passive extension of knees only to 160 degrees.

April 27, 1918. Learning to walk better with crutches. Feet in slight calcaneovalgus in walking. Knees are flexed and touch each other in walking. Braces applied.

RESULT. Was unable to walk before operation. Now walks fairly well with crutches.

No. 14. D. W., aged three years. Spastic paraplegia since birth. (Premature birth at six months; mother in labor three or four days.)

September 16, 1916. Can stand with support only. Marked right equinovalgus; left pes equinus; stands on toes. Adductors slightly spastic. Thighs can be well adducted passively. Knees can be passively extended. Eye-grounds negative.

February 20, 1917. Operation: Stoffel's, on both internal popliteals.

September 1, 1917. Has improved considerably in walking. Still walks on toes. Left foot flexes almost to 90 degrees, right foot not so well. Feet are flat.

March 9, 1918. Marked equinus. Walks on toes; heels do not touch the floor. Passive dorsiflexion not to 90 degrees. Braces ordered, to be worn four months before deciding on another operation.

September 21, 1918. Walks fairly well alone; has been improving. Contracture of both tendo Achillis. Right heel touches ground in walking; left heel does not. Requires another operation.

May 3, 1919. Able to walk with and without braces, when supported by one hand to maintain balance. Can walk without braces without assistance. No adductor spasm. Heels come to floor on walking. Moderate contracture of tendo Achillis, which can be partly overcome by stretching.

October 11, 1919. Able to walk alone. Legs and feet are in good position. Walking is a question of balance.

April 10, 1920. Patient continues to improve. Walks fairly well without braces, but with tendency to pes equinus. Is wearing braces to hold knees in extension.

June 3, 1920. Lengthening of tendo Achillis.

RESULT. Was unable to stand alone or to walk before operation. Now walks fairly well without support.

No. 15. L. V., aged three years. Spastic paraplegia, which followed some illness when four months of age.

September 23, 1916. Lower extremities very spastic. Able to stand on toes when supported; cannot walk. Mentality poor.

July 31, 1917. Operation: Stoffel's; excision of both nerves to gastrocnemii; portion of supply to solei.

May 25, 1918. Feet are straight, with no tendency to contracture of tendo Achillis. Good passive dorsiflexion, but no active motion. She is able to stand without braces flat on her feet without deformity. Knees are slightly flexed. Adductor spasticity is present.

November 7, 1918. Operation: Stoffel's, excision of anterior branches of obturators; of two nerves to the biceps, and one nerve to the semitendinosus. Dressed in plaster.

December 6, 1919. Wearing braces; walks fairly well if supported by hand. Knees are well apart, but are slightly flexed. There is no contracture of the hamstrings, the contracture is apparently in the structures of the knee-joint. Fair active dorsal and plantar flexion of the feet. Feet in good position on floor.

RESULT. Was unable to walk before operation and could stand only when supported. Now walks fairly well when supported by the hand.

No. 16. J. D., aged five years. Spastic paraplegia since two years of age.

September 15, 1917. Mother states that he has to be carried all the time, and never walks of his own accord. He is able to walk on his toes with great difficulty. Good abduction of thighs.

September 28, 1917. Operation: Stoffel's, on both internal popliteals.

November 3, 1917. Walks fairly well with legs well apart, with heels on floor.

November 8, 1919. Patient has steadily improved until he now walks fairly well alone with feet in good position on the floor, but with his knees slightly flexed. Has good active and passive dorsiflexion. Good passive abduction of thighs. No contracture of hamstrings. Braces applied to correct pes valgus.

RESULT. Walked with great difficulty before operation, now walks well with no deformity except slight tendency to pes valgus.

No. 17. L. S., spastic paraplegia. March 30, 1918. Stoffel's operation on obturators.

RESULT. Was discharged from hospital improved, and has not been seen since.

No. 18. O. T., aged twenty-three years. Spastic hemiplegia. Followed injury to head when eight years of age. Cerebral decompression was performed shortly after the injury, without results.

April 30, 1918. Her right upper extremity is very spastic. The fingers and thumb are flexed tightly into the palm. Her wrist is flexed. Her elbow cannot be fully extended. At times she has rather severe spasms in the muscles of the forearm which occasion considerable pain. She walks with a slight limp but the heel comes to the ground.

May 19, 1918. Operation: Stoffel's, on the right median nerve, resection of about one-half of the nerve supply to all the muscles of the wrist and fingers except the F. L. P. and P. Q. and the deep head of the P. R. T., with total resection of the nerve to the superficial head of the P. R. T.

August 13, 1918. Patient has experienced marked improvement. She has good active flexion and extension of wrist against gravity. Can flex fourth and fifth fingers almost to the palm, and the middle finger half way and index finger but slightly. Good active extension of thumb. Can touch thumb to little finger. Rotation of forearm about one-half normal. She is learning to write with her right hand, and is able to shake hands. She goes in swimming and her hand remains straight. The muscles of the forearm are developing.

January 1, 1919. Patient in August of 1918 became severely ill with pneumonia. During her illness the spasticity in her arm became very marked, and her fingers and hand would become strongly flexed. She had severe attacks of pain in the muscles of the forearm, but spasm was at all times more marked in the ulnar distribution than in the median. Following her recovery from pneumonia the spasm continued. Was still more marked in the ulnar distribution. The patient had lost practically all the improvement she had gained following her operation. She has continued to wear splints to support her arm but at one time had her hand corrected under an anesthetic and dressed in plaster for a time, but her improvement has been slow. She did not begin to get return of voluntary motion of the fingers and wrist for about six months, and she has not yet returned to her condition preceding her illness.

RESULT. This patient had marked improvement in the appearance and function of her hand following the operation, and then suffered a severe relapse which was due to an attack of pneumonia.

No. 19. H. A., aged five years. Spastic diplegia since one year of age.

July 8, 1918. Neither talks nor walks. Mentality feeble. She is able to stand on her feet when supported. Knees are slightly contracted. Crosses her legs when standing. Feet are in a position of calcaneovalgus.

July 10, 1918. Operation: Stoffel's, on anterior obturators, double subastragalar arthrodesis, and forcible extension of knees.

August 8, 1918. Discharged from hospital and walking on casts.

RESULT. Improved.

No. 20. W. L., aged four years. Spastic paraplegia since eighteen months of age.

July 20, 1918. Right elbow is held flexed. Good power in biceps and triceps. Arm is pronated and there is good power in the grasp and almost normal active extension of wrist and fingers. Drags right leg in walking. Active supination 45 degrees beyond mid-position. He walks on the toes of the right foot. Slight contracture at the knee. Good adduction of thigh.

August 13, 1918. Operation: Stoffel's on internal popliteal.

May 3, 1919. Walks very much better with foot flat on the floor and does not drag toes. Active dorsiflexion of foot to normal extent. No deformity of foot. Fair function of right hand and arm.

RESULT. Fair improvement.

No. 21. C. W., aged nine years. Spastic paraplegia. Mental deficiency. Does not talk.

July 27, 1914. Cannot walk alone. Stands on toes with knees together.

August 19, 1918. Is learning to talk. Is able to stand when supported by hand. Contracture of both tendo Achillis. Left pes valgus on standing.

August 28, 1918. Operation: Stoffel's, on anterior obturators, lengthening of both tendo Achillis.

December 9, 1918. Braces have been applied, legs are in good position with thighs well apart and knees straight. Patient is learning to walk.

RESULT. Moderate improvement.

No. 22. A. S., aged twenty-two years. Spastic paraplegia. Had severe paraplegia and had a Foerster operation three or four years ago. As a result of this operation she was greatly improved. Walking much better, with her feet in fair position with rather marked adductor spasm so that her knees would rub together in walking.

January 14, 1919. Operation: Stoffel's, on both anterior obturators. Patient was discharged from hospital about two months later and her walking was very greatly improved. Have been unable to follow this case since operation.

RESULT. Very much improved.

No. 23. D. M., aged five years. Spastic paraplegia, congenital.

January 11, 1919. Has never walked; is able to stand on his right leg, but not on his left. Left knee is held flexed. Both tendo Achillis contracted. Marked double pes valgus. Adductors not spastic. Has marked scoliosis.

January 30, 1919. Operation: Stoffel's, on both anterior obturators, both branches to gastrocnemius and almost all of bundle to soleus. No contracture of tendo Achillis under ether. Dressed in plaster from pelvis to toes.

February 8, 1919. Lateral traction to spine begun.

August 16, 1919. Discharged from hospital, wearing spinal brace and thigh brace with lock-joint at knees. Able to walk when held by hand. Knees are flexed unless held extended by braces. Practically normal active dorsal and plantar flexion of feet.

June 3, 1920. Walks fairly well without support.

RESULT. Marked improvement.

No. 24. J. G. Spastic hemiplegia. March 22, 1919. Marked spasticity of left arm and left leg. Able to walk only if held by hands; walks on toes of left foot.

April 3, 1919. Operation: Stoffel's, on one popliteal.

July 5, 1919. Discharged from hospital walking very much better. Heel comes to ground. Walks without assistance.

RESULT. Marked improvement.

No. 25. A. M., aged twenty-six years. Spastic paralysis, spinal. March 25, 1919. Operation on both obturators and popliteals. (Report given by a neurologist, Dr. W. B. Cadwalader.) "A. M.'s paraplegia is organic in character, caused by a chronic degeneration of the pyramidal tracts of the spinal cord, which, I think, may have followed an infection, but is not syphilitic. The condition has existed for about five years and has progressed but very slowly. She is very neurotic and has had many hysterical symptoms from time to time. Her only chance will be what you can do, such as a tenotomy, this should help her a lot."

February 23, 1920. When discharged from hospital she was greatly improved.

RESULT. This case has not been followed but showed marked improvement when last seen.¹

No. 26. T. F. Spastic paraplegia; right arm and both legs. Followed illness when small child.

April 16, 1919. Operation: Stoffel's, on both obturators, and tenotomy of left adductor; also tenotomy of right hamstrings.

July 16, 1919. Application of cast to fractured femur.

RESULT. Case not followed.

No. 27. E. P., aged nine years. Spastic paralysis. March 18, 1919. Walks with spastic "scissors" gait, on toes, and with crossing of knees. Moderate spasticity. Ankle and patellar clonus marked. Both legs stiff. Can bend knees when legs are flexed on the thigh, but not if extended. No Babinski.

May 22, 1919. Operation: Stoffel's, on both obturators and popliteals.

October 11, 1919. She has been discharged. She can walk by herself only with the aid of a cane or when held by one hand.

May 8, 1920. Walks readily a half mile without assistance.

RESULT. Moderate improvement.

No. 28. G. P., aged seven years. Spastic paralysis. March 18, 1919. Cannot get out of bed by himself, unless very awkwardly. Feet are flexed, adductors spastic. All reflexes exaggerated. Patellar and ankle clonus. Babinski positive both sides. Walks with the aid of a cane and when supported by hand.

May 22, 1919. Operation: Stoffel's, on both obturators and both popliteals.

October 11, 1919. Discharged. Walks fairly well by himself, with feet flat on the floor and without crossing of legs.

RESULT. Marked improvement.

No. 29. S. A. Spastic paralysis. June 26, 1919. Operation: Stoffel's, on both popliteals and obturator nerve.

February 21, 1919. Discharged from hospital improved, but has not been seen since.

RESULT. Moderate improvement.

¹ February 8, 1921. This patient has been seen a number of times recently. She states that preceding the operation she had been unable to work for five years and scarcely able to walk for three years. She returned to work a month after leaving the hospital, and has worked steadily ever since. She walks almost as well as she ever did, except, when becoming excited, her gait becomes slightly spastic.

No. 30. A. S. *Spastic paralegia. July 14, 1919. Parents and three brothers and one sister living and well. Has had paraplegia for three or four years. Stoffel operation four weeks ago; excision of anterior branches of both obturators and nerves to both heads of right gastrocnemius and portion of branch to soleus.*

August 11, 1919. Walks better than formerly. Heels to ground and knees slightly apart. Can abduct legs fairly well. Active dorsiflexion of right foot to behind 90 degrees.

November 24, 1919. Heels do not quite touch ground in walking, due to holding knees flexed. Legs internally rotated. Good passive abduction of thighs.

RESULT. Moderate improvement.

No. 31. M. P., aged eight years. July 14, 1919. *Spastic paralegia. Premature birth (between seven and eight months). Cannot talk, but is able to copy writing or printing. Able to walk with great difficulty with accelerated gait; walking on toes and crossing legs. Heels do not come to floor.*

July 16, 1919. Operation: Stoffel's, on both internal popliteals and anterior obturator nerves. Wounds healed except for small spot in both popliteal incisions. Is already walking better. Referred to nose and throat specialist dispensary for examination for mutism.

December 1, 1919. Heels come to ground in walking. Legs well apart. Gait is markedly improved. Drags toes on walking. Swings legs to side on walking, instead of moving them forward. Active dorsiflexion almost to right angle.

RESULT. Marked improvement.

No. 32. D. B. *Spastic paralysis. December 4, 1919. Operation: Stoffel's, resection of the anterior branches of both obturators, and of both nerves to the gastrocnemius, one nerve to the soleus and a portion of the nerve to the right tibialis posticus.*

Child was able to stand and to walk when held by the hand, but she stood on extreme tiptoes with the right foot slightly inverted and the left foot slightly everted. At times her knees would strike together. Mentality seems fair. Her thighs could be separated about one-half normal.

December 6, 1919. Good active dorsiflexion of left foot to beyond right angle and of right foot to about right angle. Good active plantar flexion.

February 23, 1920. Pads have been placed in her shoes to correct

the tendency of pes valgus. She walks quite well without assistance. Heels on floor; knees do not strike.

RESULT. Marked improvement.

CONCLUSION. Thirty-two cases have been operated on by the Stoffel method. They have been under observation for periods of time up to six years. No definite relapse has occurred in any. The youngest patient was three years of age, the oldest twenty-seven. Fifteen cases were congenital in origin, 11 infantile, 4 traumatic, and 2 were due to spinal degeneration in adults.

No ill results followed operation in any one. Two cases required a second operation. The operations on the popliteal nerve have been almost uniformly successful in relieving the contracture of the tendo Achillis and in enabling the patient to stand and walk with the foot firmly on the floor and in greatly improving the gait. The operations on the obturator nerve have in all instances corrected the adductor spasticity, which in most of them interfered greatly with walking. The results of the operations on the sciatic nerve for hamstring contracture have also been good. The operations on the median nerve have in all instances lessened the spasticity or corrected the deformity of the hand. In some instances the improvement in function of the hand was slight or negligible; in other instances it was very great. The hand is such a complex mechanism that it is more difficult to secure return of function when there is marked spasticity, and I feel that the results to be secured here are more uncertain than those to be secured in the lower extremity. The improvement is sometimes more brilliant, but I do not feel so confident of it as I do of the results to be obtained in the lower extremity.

Where the patients have been of good mentality the results have always been very much better than where the patients have been backward or feeble-minded. I believe that we may always count on being able to cure any contractures that may be present whether in the upper or lower extremity, and the spasticity may be greatly relieved, but the amount of function, that is, of voluntary active motion of the member, which the patient will secure depends upon the nature and

the severity of the disease, upon the mentality of the patient and upon the careful, persistent after-treatment. In certain cases the deformity is relieved and the spasticity is improved, but the patient is unable to establish voluntary control of the member. This, I take it, is possibly due to the organic nature of the disease. In such cases the cerebral control has been definitely and completely lost, either by injury to or disease of the cerebral centers or the cord. Even in such cases the patients are more or less improved by the moderation of the spasticity in the lower arc.

These methods of treatment have been applied to all spastic cases under my observation within the past six years. I believe that the Stoffel procedure is the best single operation which has thus far been proposed, because it is applicable to a greater number of cases, the results obtained are more uniformly successful, the operation itself is in no way a severe one and no ill results appear to follow it. Tendon lengthening is required in some cases and transplantation may be suitable in others; both of which may be combined with the Stoffel operation. It is not to be concluded that the Stoffel neurectomy is a panacea for spastic paralysis, nor that it is indicated in all cases. Judgment must be used in the selection of cases and in the time and application of the operation.

INFECTIOUS OSTEOMYELITIS: DIFFERENTIAL DIAGNOSIS*

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THE interpretation of roentgenograms depends, to a large extent, upon the degree of knowledge possessed by the interpreter of the pathological changes which are shown on the roentgenograms. Lovett and Wolbach¹ have pointed out that pathological conditions in bone give rise to only three processes: that of (1) bone proliferation and new bone formation; (2) rarefaction or bone destruction and (3) bone atrophy or loss of calcium salts. The rarefaction may be limited or widespread, the bone production may be confined to the bone itself or, as in the malignant lesions, it may invade the surrounding tissues. In some instances cysts may develop in bone and they may be caused by a variety of diseases. It is very unusual for only one of these processes to be present, but often combination of only two of them may result. Long since, a definite conformation and appearance of these pathological changes on the roentgenogram have been described as typical of infectious or pyogenic osteomyelitis and have served as the criteria of differentiation from other diseases.

But there have arisen in the study of this disease, as of any other, variant or transitional types of roentgenographic changes which are very difficult to recognize and differentiate from other disease types and indeed at times they may even more resemble the accepted typical picture of some other disease than of osteomyelitis itself. The illustration and study of these types is the excuse for the presentation of this paper.

In approaching the subject of cystic disease of bone, which

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FIG. 1

FIG. 2

FIG. 1.—Osteomyelitis. Areas of rarefaction in the distal extremity of the shaft and the lower epiphysis of the femur. Culture of pus obtained at operation, *Streptococcus hemolyticus*. The absence of atrophy of the shaft and the comparatively short duration of symptoms prior to roentgen examination aid in the differentiation from tuberculosis.

FIG. 2.—Large Brodie's abscess of femur. Culture of pus *Staphylococcus aureus*. Well-marked periosteal reaction.

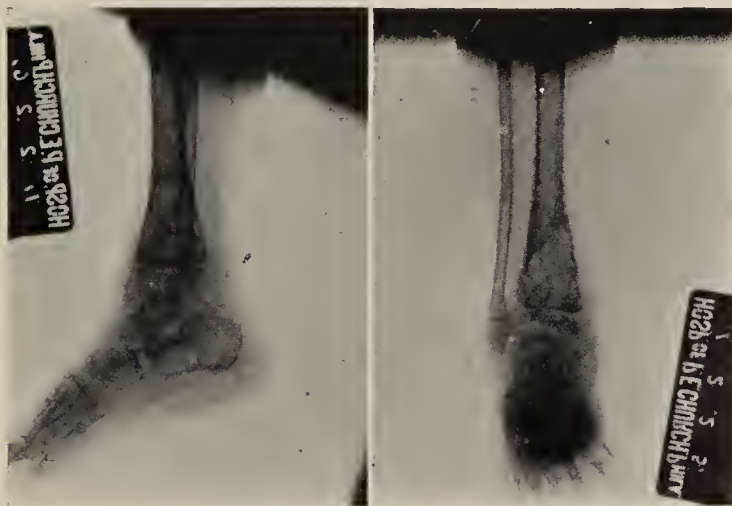


FIG. 3.—Wedge-shaped area of rarefaction in the lower extremity of the diaphysis of the tibia. Duration two years previous to roentgen examination. At operation the periosteum was found to be slightly thickened and the cortex was slightly hyperemic. The cortex was about 2 mm. thick, and when this was removed a yellowish-white cyst-wall bulged into the opening. When this was cut through with scissors, some semifluid matter resembling tuberculous pus oozed out, and then considerable cheesy matter was evacuated, but no free fluid. The cyst cavity extended down to the epiphyseal cartilage. Culture and smear from the cyst were negative. The cyst-wall and contents did not reach the laboratory in fit condition for histologic study or for inoculation into guinea-pigs.



FIG. 4.—Osteomyelitis. Culture of pneumococcus obtained from pus at operation. Smears of pus negative for tubercle bacilli. The rarefied area in the lower end of the diaphysis of the tibia is wedge-shaped, with the base toward the epiphyseal line, surrounded on the side of the shaft by increased density and inflammatory reaction; moderate involvement of the periosteum.

may be caused by a variety of pathological conditions, Ashhurst, Bromer and White² have classified all diseases of bone under three general categories: infections, dystrophies and tumors. It has seemed best to approach the differential diagnosis of pyogenic osteomyelitis in the same manner, illustrating, first, cases of other infections with which it may be confused; then cases of tumors and then of dystrophies.

Of the infections no definition is necessary; syphilis and tuberculosis are the two most likely to cause confusion. Ashhurst² defined the dystrophies as (1) remote or extremely attenuated infections from which no causative organism can be obtained, or (2) as alterations or congenital abnormalities of metabolism whose true cause may or may not be determined. The term tumor was used in the clinical sense, inasmuch as the pathological and pathogenical sense of the term is still in dispute. A tumor may be an embryonal (malignant) type, an intermediary (questionable) type or an adult (benign) type, there being thus many tumors which, as Ewing says, are "neither benign or malignant." There are also many tumors which are neither true granulation tissue (inflammatory reaction) nor yet true tumors. It is this transition from inflammatory reaction to true tumor growth which makes the differential diagnosis of many lesions not only difficult but almost impossible both for the roentgenologist and the pathologist.

GROUP I, INFECTIONS. Pyogenic infections of bone cause, first, destruction of bone, followed by repair, the destruction predominating in the acute stage before systemic reaction is sufficient to start reparative and formative changes. The destruction means necrosis of bone, the formation of sequestra with new bone or involucrum surrounding the individual sequestrum. When this proceeds in the normal manner it gives the clinical roentgen picture by which pyogenic osteomyelitis is diagnosed and differentiated on the roentgenogram. But often, instead of sequestration, there is hardening and thickening of bone with a comparatively small central area of destruction. These cases take the form of Brodie's abscess, or Markoe's abscess, or the condition described by Garré: merely bone sclerosis without any demonstrable pus or central cavity.

Thus the usual description of osteomyelitis is first bone destruction, followed by repair and new bone formation. Tuberculosis is regarded as usually destructive, occurring near the epiphysis (Fig. 1), and characterized by marked atrophy of the shaft of the affected bone; syphilis is considered as largely formative or proliferative, with comparatively little or no bone destruction.

Tuberculosis. Tuberculosis and pyogenic osteomyelitis can produce similar roentgen appearances. According to Lovett and Wolbach,¹ first, the so-called Brodie's abscess (Fig. 2) can be simulated by both diseases; second, wedge-shaped areas of rarefied bone (Figs. 3 and 4), with varied degrees of cortical thickening, with their bases toward the articular extremities of the bones—can be found in both conditions; third, they make mention of the occurrence of tuberculosis of the shaft, resulting in a picture very much like that of osteomyelitis. There are probably more case reports of such occurrence in Continental and English journals than in the American. Brown and Stiefel³ have reported one case with a roentgen appearance of the multiple lesions which made a differential diagnosis between syphilis and tuberculosis very difficult. At no time, however, was the diagnosis of osteomyelitis of pyogenic origin considered a possibility. The diagnosis of tuberculosis was verified by pathological and autopsy findings. Three of the cases reported by Lovett and Wolbach had definite involvement of shaft, with little or no extension to the epiphyseal line, and the roentgenograms resemble osteomyelitis. Allison and Fisher⁴ were able to produce tuberculosis experimentally in any portion of the long bones of dogs. They found that when the focus was implanted under the periosteum or in the diaphysis, a proliferative process, resulting in the formation of new bone, occurred. When the epiphysis or the joint surface was inoculated this reaction did not occur. This observation seemed to bear out the clinical classification of John Fraser (*Tuberculosis of Bone and Joints*, 1914) in that he described a hypertrophic type of bone tuberculosis which occurs in the shaft of long bones. In six years at the Episcopal, Children's and Orthopedic Hospitals only one case of this type of tuberculous

involvement has been noted, and because of the rarity of this occurrence a full report of this case is appended.

Phemister⁵ has recently called attention to the differential diagnosis of pyogenic and tuberculous arthritis. He points out that pyogenic arthritis quickly destroys cartilage and, if the infection is severe, the articular cortex on joint surfaces where weight-bearing is exerted. In tuberculous arthritis the cartilage has a tendency to remain intact for a long time between weight-bearing surfaces and it is destroyed first by the direct attack of granulation tissue along the free surfaces. It stands longest in the regions of contact and greatest pressure in the joint. Consequently, the joint cleft, or clear space between articulating bones on the roentgenogram, is preserved for a longer time in tuberculous arthritis. He further states that secondary invasion of the bone, resulting in sequestra bordering on the articular surface, is rare in primary pyogenic arthritis, but is common as a late occurrence in tuberculous arthritis. However, if the pyogenic infection is primary in the shaft, and later extends into the epiphysis and the joint, necrosis and sequestration bordering on the joint sometimes takes place. This might lead to a picture that could easily be confused with cases of tuberculous arthritis in which sequestration bordering on the joint has occurred. In two or three weeks' time the cartilage in these pyogenic cases is often destroyed by proteolytic action of the pus, hence there should be a diminution of the width of the joint cleft and this should be of assistance in making the diagnosis. Another point of aid should be the comparatively early appearance of the sequestrum in osteomyelitis, in contrast to the late appearance in tuberculosis (Fig. 5).

As a result of the study of the cases of this series the commonly accepted sign of atrophy of the affected bone, with perhaps atrophy of contiguous bones of the affected limb, can still be regarded as a fairly constant finding in many cases of tuberculosis. Likewise the statement of Codman,⁶ that in the early stages of tuberculosis in children the centers of ossification of the affected epiphyses are enlarged and rectangular instead of pyramidal, can also be so regarded. This has been called the "squaring of the epiphyses."

Syphilis. According to Lovett and Wolbach,¹ syphilis affects bone in two ways: (1) There are local, rapidly formed gummatous lesions causing destruction of bone. They may be endosteal or periosteal or perivascular, and they may extend into the cortex and medullary canal. (2) Syphilis may only slightly affect the periosteum and endosteum, and, as a result of developing osteoblasts, new bone is formed. In the first way, the bone is choked by the gummatous process, and in the second, there is only stimulation of bone-forming tissue, and the result is in the main a proliferative and formative process.

Wilhelm,⁷ in his work on the differentiation of syphilis and osteitis fibrosa, states that all congenital and acquired syphilitic changes of bone are the result of two fundamental processes going on simultaneously: First, the destruction of bone substance by syphilitic granulation tissue, and second, the new formation of bone. Added to this are changes due to complicating infiltrative and ulcerative processes in the overlying soft parts, occurring in superficial bone lesions, together with secondary reparative formation of new bone around gummata. As a result, many variations are seen in the roentgenological picture of bone and joint syphilis.

The proliferative or hyperostotic form of syphilis, as he calls it, shows (1) marked periosteal involvement and participation in the new bone formation; (2) more or less narrowing of the medullary canal, and (3) new bone laid down parallel to the long axis of the shaft. All roentgenologists will agree that these changes can be seen in the chronic form of osteomyelitis when the process has become almost, if not completely, a formative one. In these cases dependence must be placed on the history, the Wassermann reaction and sometimes on the therapeutic test. A fusiform shape of the shaft can occur in both conditions.

Again, it has been the experience of many to see the early periosteal change in young adults which cannot be differentiated by the roentgen-ray alone. An acute pyogenic periostitis will often resemble that of syphilis and the symptoms of acute inflammation may not be marked. Baetjer and Waters⁸ believe that if the lesions are multiple, accompanied by an osteitis without a cavity, the lesion is most likely to be

syphilis. They describe the "lace work" type of periostitis in which the calcium salts are laid down perpendicular to the shaft, extending in a band about a quarter of an inch from the shaft, often for half its length or more, and regard this as invariably syphilitic in origin. But this type is not often encountered. Their main reliance on differentiation is the fact that extensive bone destruction is not accompanied by the symptoms of inflammation which would naturally occur if the changes were due to an acute infection.

In the congenital syphilis which is so often a joint lesion, experience in the cases studied at the Children's Hospital has shown that in almost all cases with joint lesions the characteristics usually described as typical of this condition were found, namely, the presence of a well-developed periostitis, the preservation of the joint without cartilaginous change, and the occurrence in the first few years of life. Single lesions have been encountered as often as multiple. Baetjer and Waters state that rarefied, punched-out areas are often seen in the diaphyseal ends. These areas should not be confused with rarefied areas sometimes seen in osteomyelitis situated near the diaphyseal ends (Figs. 6 and 7). In cases of early osteomyelitis the clinical history has helped to differentiate it from syphilis. Granting that tuberculosis of the shaft does occur, its differentiation from syphilis seems to be a more difficult matter than the differentiation of osteomyelitis from syphilis.

GROUP 2. TUMORS. Probably the two most likely tumors with which osteomyelitis might be confused are certain types of osteogenic sarcoma and endothelioma of bone (Ewing's tumor). Occasionally the early destructive stage of osteomyelitis might resemble a metastatic carcinoma of bone when it occurs in a patient of the age in which carcinoma is most often seen.

There are two types of osteogenic sarcoma which might be difficult to differentiate from osteomyelitis. The first is the very early case of the type formerly spoken of as periosteal sarcoma. Here the term periosteal sarcoma will be used in its older sense, inasmuch as it was previously so called to the exclusion of any other name, before the new nomenclature

of the Bone Registry made its appearance. At no time will it be used to designate the fibrosarcoma, juxtaperiosteal, as is the case in the nomenclature of the Registry, for which Ashhurst prefers the old name parosteal as being more definitive. In its well-developed stages, the periosteal (osteogenic) sarcoma should not be mistaken for osteomyelitis; but in its early stage (Fig. 8), when practically the only sign is bone destruction with invasion of the cortex, with no demonstrable invasion of the surrounding soft parts, the picture is very likely to be confusing. Of diagnostic aid is the point of differentiation made by Baetjer, *i. e.*, osteomyelitis has a tendency to destroy cortex in somewhat scattered areas, sarcoma tends to destroy it *en masse*, with no intervening scattered areas of normal cortex. The well-known perpendicular striations of new bone growth cannot always be found in the early stage and characteristic "sun-ray" form is sometimes entirely lacking.

Indeed, it seems that absolute dependence cannot be placed on the sign of new bone laid down perpendicular to the shaft. In 2 of the cases of osteomyelitis of this series such striations are present. In the first (Figs. 9 and 10) the clinical history quite definitely established the diagnosis; in the second, no absolute diagnosis could be made before operation.

The second case (Fig. 11) was that of an adult, aged thirty-nine years, with a history of deep bone pain in the lower third of the right tibia for three weeks previous to the first roentgen examination. The elevation of temperature never exceeded 100° , rarely reaching that point. There was a history of perinephric abscess six years previous, and of a nasal accessory sinus infection six months previous. The first roentgenogram showed a fusiform periosteal and cortical enlargement very suggestive of syphilis. The Wassermann reaction was negative. A simple incision was made, cutting down merely through the periosteum (the intention being to so relieve the deep bone pain), and was closed under aseptic precautions. No pus was encountered. The severe pain persisted with no more elevation of temperature than previous to the operation, and four weeks later another roentgen examination showed the same fusiform swelling with definite perpendicular striations.

Exploratory operation, using an Esmarch bandage above the knee as a precautionary measure, exposed a deep collection of pus within the cortex and medullary cavity from which a culture of staphylococcus was obtained. The question naturally arises, did the first operation, having been surgical interference with the periosteum, give rise to these striations? Against this supposition is the fact that syphilis also has been known to produce the same change (Pancoast, verbal communication) (Baetjer and Waters).

The second type is that of the so-called sclerosing type of bone sarcoma. When the tumor has produced dense bone it can resemble the chronic type of osteomyelitis without sequestration. Sometimes these may show a variable amount of periosteal proliferation. Codman regards this diagnosis as rather easy.

When a lesion presenting periosteal change on the roentgenogram is seen, it is well to recall Bloodgood's⁹ classification of periostitis in order to include all possible causes:

1. Traumatic ossifying periostitis.
2. Syphilitic ossifying periostitis.
3. Pyogenic ossifying periostitis (associated with osteomyelitis).
4. Ossifying periostitis about tuberculous lesions of bone.
5. Ossifying periostitis associated with benign tumors of bone.

In syphilitic ossifying periostitis he states that if lesions are multiple, sarcoma can be excluded. The Wassermann reaction and specific treatment will establish the diagnosis. In pyogenic ossifying periostitis it is rare to see the periosteum affected without cortical change except in the case of gonorrheal infections, usually found in the os calcis. Multiplicity of lesions, of course, aids in the diagnosis of osteomyelitis. Bloodgood has had no cases of tuberculous involvement of the bone previously diagnosed as sarcoma.

Ewing's Tumor. Solitary diffuse endothelioma of bone has been mistaken for osteomyelitis. Ewing¹⁰ states that this is a relatively frequent and quite specific disease which has often been unrecognized and has passed for round-cell sarcoma of bone. It seems best to give his own description of this condition:

"Most of the cases that I have seen have been in young subjects—3 of them fourteen years of age, the oldest nineteen years. The disease begins without assignable cause, with attacks of pain aggravated by exercise, but not very severe, so that the patient is able to go about for weeks or months. A spontaneous fracture may occur early. A diffuse swelling slowly forms over the affected bone and a tumor tissue involves the soft parts. Fluctuations in the size of the tumor may then be noted. The growth is comparatively slow, several months elapsing before the external swelling demands attention. At this time the radiograph shows a smooth, diffuse rarefaction of a large portion of the shaft of the long bone, the outlines of which are widened. The location in the shaft rather than at the end of the bone and the absence of bone production distinguish the condition from solid osteogenic sarcoma, while the failure of early and sharply limited bone destruction is contrasted with the radiograph of telangiectatic bone sarcoma."

He makes no mention of the confusion of this disease with osteomyelitis, but instances of this sort have occurred and these tumors have been excised for diagnosis or treatment and have even been treated for a considerable length of time as osteomyelitis. They respond rapidly to roentgen-ray and radium treatment, but the ultimate outcome is fatal. This regression under irradiation can be of diagnostic assistance. Codman¹¹ emphasizes the fact that this tumor may be multiple, that it shows a characteristic longitudinal striation and that it involves more than half the shaft. It does not produce radiating spicules, but produces onion-like layers of periosteal new bone formation such as seen in osteomyelitis. He also states that the roentgenological appearance is usually confused with osteomyelitis. It is an invasive, bone-destroying tumor, but can produce reactive new bone growth as does bacterial infection.

No instances of this tumor have been studied for this work, as none have occurred in six years on any of the services of the Episcopal, Orthopedic or Children's Hospitals. This, however, should not be regarded as evidence of the infrequency of this disease. The possibility of its occurrence must always be remembered and especial effort made not to pass upon it as an osteomyelitis.



FIG. 5.—Pyogenic osteomyelitis and arthritis of the hip-joint in which the infection was primary in the shaft. A portion of the epiphysis of the head of the femur has become sequestrated. The joint space is decreased in width, owing to the destruction of the joint cartilage; there is marked atrophy of the shaft of the femur resembling that found in many cases of tuberculous arthritis. Culture of pus obtained at operation, *Staphylococcus aureus*.



FIG. 6.—Aged six weeks. Blood Wassermann negative; von Pirquet negative. Pus from abscess at operation, staphylococcus. Two small sequestra can be seen, otherwise the roentgenogram resembles syphilis.



FIG. 7.—Aged four months. Congenital syphilis involving the periosteum of the distal extremity of the diaphysis of the humerus with preservation of the epiphysis of the capitellum. The usual type of joint lesion in syphilis. Multiple lesions. Blood Wassermann: cholesterin, positive, 4 +; acetone, positive, 4 +.



FIG. 8.—Periosteal sarcoma of the tibia, erroneously diagnosed at first as acute osteomyelitis. Symptoms first noted following traumatism to leg, three weeks before this roentgen examination. Eight weeks later, examination showed perpendicular striations and an appearance typical of a periosteal sarcoma.



FIG. 9.—Chronic osteomyelitis; duration thirteen months previous to roentgen examination. Culture from operative wound, *Staphylococcus aureus*. Blood Wassermann, twice negative. Pathological diagnosis: chronic suppurative osteomyelitis with fibrous tissue. Guinea-pig inoculation, negative. Perpendicular striations and sequestra are present.



FIG. 10.—Chronic osteomyelitis; duration thirteen months previous to roentgen examination. Culture from operative wound, *Staphylococcus aureus*. Blood Wassermann, twice negative. Pathological diagnosis: chronic suppurative osteomyelitis with fibrous tissue. Guinea-pig inoculation, negative. Perpendicular striations and sequestra are present.



FIG. 11.—Osteomyelitis, four weeks after incision of periosteum, without drainage, for relief of pain, no pus having been found. Small fine perpendicular striations are present. On the day following this roentgen examination, pus was obtained from a deep bone abscess from which a culture of staphylococcus was obtained. Blood Wassermann, negative.



FIG. 12.—Acute osteomyelitis. The process is entirely destructive, with a pathological fracture. The roentgen appearance resembles metastatic carcinoma. Severe infection of the hand six weeks previous. Because of this definite clinical history there was no difficulty in differential diagnosis.

Metastatic Carcinoma. Occasionally during the rarefied, early stage of osteomyelitis, with a very ragged appearance of the periosteum, the appearance may resemble that of metastatic carcinoma. In the one case of osteomyelitis in this series presenting such change, with a pathological fracture, the diagnosis was made certain by the history of previous severe infection of the hand and forearm, the lesion occurring in the upper third of the humerus. It would seem that in all cases, even if the roentgen changes are deceptive, the two diseases should not be confounded because of the history and clinical signs and symptoms (Fig. 12).

GROUP 3. DYSTROPHIES. The necessity for differential diagnosis between infantile scurvy and the early stage of acute osteomyelitis and epiphysitis is by no means infrequent. The clinical picture of a fretful, miserable child with swollen (sometimes reddened) joint, marked pain and tenderness on movement and elevation of temperature, is apt to be confusing. The roentgen-ray can be of great service in diagnosis. If it is osteomyelitis in the very early stage, there will most likely be no demonstrable bone changes on the roentgenogram. If it is a later stage, periosteal change with some rarefaction and destruction of the cortex may be found. In all cases in making this diagnosis, the early changes of scurvy must be sought. As Pelkan has shown, it is not necessary that subperiosteal hemorrhage be present to make the diagnosis of scorbutus. In the early stage, the one most likely to be confused with osteomyelitis, the following are the roentgenographic signs which establish the diagnosis: (1) The dense ringlike shadow of the calcified matrix of the border of the epiphysis, with the smooth, ground-glass appearance of its center (Wimberger¹²); (2) the smooth, ground-glass appearance of the shaft of the affected bone; (3) the calcified matrix of the distal end of the diaphysis, *i. e.*, the epiphyseal line; (4) if the disease is more advanced, there will be found the rarefied area either just above or below the calcified epiphyseal line, called by Pelkan¹³ "the scurvy line." These last two signs have been indiscriminately spoken of as the "white line" of scurvy and the Trümmerfoldzone or the Trümmerzone. The white line (calcified epiphyseal line) has been proven not to be pathog-

nomonic of scurvy (Lovett¹⁴), but Pelkan claims the "scurvy line" can be so considered. Finally, the last sign (5) can be looked for, the so-called spurs (projections extending outward at the diaphyseal ends which are the first signs of subperiosteal hemorrhage and are due to the shoving out of the zone of preparatory calcification which is greatly weakened in scurvy). Superadded trauma will easily produce epiphyseal separations, another diagnostic point. Since some of these signs will always be found in combination of at least two or more, this differential diagnosis can be considered a rather easy task (Fig. 13).

Osteitis Fibrosa Cystica. Due to the fact that osteomyelitis can cause changes in bone simulating the appearance of bone cysts, and due to the fact that occasionally these rarefied cystic areas may be multiple in character, difficulty may arise in its differentiation from the disease entity known as osteitis fibrosa cystica, or chronic cystic osteitis. Under this head will be considered both localized cystic disease and also the general type usually coupled with the name of von Recklinghausen. According to Ashhurst's classification, cysts due to infections were discussed under the first group. If the inflammatory theory of the causation of this condition is accepted, then it, too, should have been there discussed. The theory, however, of metabolic disturbance as the cause of von Recklinghausen's disease has never been disproved, and it has seemed more logical to place both the localized and general types under the head of dystrophies, inasmuch as, in general, the pathological characteristics of each are very similar. It should be added, however, that to the three primary pathological bone manifestations in all diseases of bone, a fourth might be added for this disease, that of fibrous-tissue substitution. The areas of lessened density are caused in part by the replacement of osseous tissue by fibrous-tissue growth.

Localized chronic cystic osteitis probably occurs more often in the growing periods of life, but 2 of the cases studied in this series occurred during the third decade. The usual sites for its appearance are the proximal ends of the femur, humerus and fibula, but cysts of the middle third of the shaft are by no means rare. The cortex is intact and has a tendency to

expand in a fusiform or cylindrical manner. The area of rarefaction is sharply limited and has a definite outline showing no invasion of the soft tissues. Cystic areas of osteomyelitis, on the other hand, are apt to have inflammatory thickening of the cortex and periosteum in the neighboring areas. A thorough search should always be made for any signs of a sequestrum. In osteomyelitis trabeculations are usually absent, while in cystic disease they are often present.

It is possible, too, that transitional types can exist in the histological picture. Thus some areas in bone cysts which do not have the usual inner fibrous-tissue lining may have the appearance of the inflammatory tissue that results from infection by the usual bacterial agents. In osteomyelitis due to an extremely attenuated and chronic bacterial infection there may be no pus within the cystic cavity, but instead only a hemorrhagic content (Fig. 14). In addition, on the roentgenogram the inflammatory zone of increased density may be slight or almost entirely absent. In progressing a step further in like fashion, there are transitional types grading from the bone cyst to the giant-cell tumor, due to the presence of a greater number of the epulis type of foreign-body giant cells, resulting in a more or less confusing appearance on the microscopic slide. In some of these cases it is absolutely impossible to differentiate a bone cyst from a giant-cell tumor by the roentgenogram. The ultimate dependence in the differentiation between cystic change in osteomyelitis and chronic cystic osteitis is upon the extent of evidence of inflammatory manifestations: thickened cortex, periosteal proliferation and sequestration.

Generalized fibrocystic disease presents a very typical roentgenogram. Several long bones are usually affected, marked deformity due to bowing occurs, and the cortex is expanded and thinned. Within the medullary cavity are large and numerous rarefied areas. The normal bone detail is replaced by irregular strands of trabeculæ. Baetjer cites a case with periosteal proliferation. As in osteomyelitis, there is sometimes actual lengthening of the bone. It seems that this, combined with the clinical picture, should give no difficulty in the differential diagnosis. The problem is not so

much that of differentiation from osteomyelitis as it is that of differentiation from syphilis.

CONCLUSIONS. When pyogenic osteomyelitis conforms to its usual pathological manifestations, it will produce a roentgenogram which will usually be diagnosed without error.

When it undergoes the destructive stage followed by the proliferative stage, it probably will not be confused with the other infections, syphilis and tuberculosis, especially when it is accompanied by large and well-defined sequestra. Syphilis will be usually formative and tuberculosis will be largely destructive, combined with bone atrophy. The chronic stage of osteomyelitis, when the picture is that of a formative process and no evidence of sequestration and involucrum formation can be found, is likely to be confused with syphilis, and the differentiation depends largely on the history and the laboratory findings. Tuberculosis of the shaft, although rare in occurrence, will probably only be differentiated by other means than the roentgen-ray alone. To the three special forms of manifestations of osteomyelitis and tuberculosis, mentioned by Lovett and Wolbach, which may cause similar roentgen appearances, a fourth (Phemister) is added.

Ewing's tumor, or endothelioma of bone, is most likely to be confused with osteomyelitis inasmuch as the usual criteria of malignancy are not to be seen (Codman). On the history must be placed most dependence. The early stage of periosteal sarcoma cannot always be differentiated because occasionally in these cases pyrexia, pain and swelling may simulate the signs and symptoms of acute osteomyelitis.

Too much dependence must not be placed on the occurrence of perpendicular striations as a pathognomonic sign of malignancy. These have been noted in cases of both acute osteomyelitis and syphilis.

Of the dystrophies, infantile scurvy will cause no difficulty if its early signs are sought for and found on the roentgenogram. Bone cysts will be differentiated with fair degree of accuracy if the signs of pyogenic inflammatory change are recognized. In cases of osteomyelitis with hemorrhagic content in the rarefied area (abscess) of bone, and with no definite sequestrum, due to attenuated bacterial infection,



FIG. 13.—Illustrates the changes of early scurvy: the dense rings surrounding the epiphyses, the ground-glass atrophy of the shafts, the dense lines at the diaphyseal ends. The "scurvy line" is as yet scarcely defined.



FIG. 14.—Chronic osteomyelitis. First symptoms were noted two years before roentgen examination. No pus found at operation, only hemorrhagic fluid. Culture from this fluid showed *Staphylococcus aureus*. Pathological diagnosis of bone tissue removed at operation, chronic inflammatory tissue. The inflammatory zone of increased density surrounding the rarefied area and the poorly defined sequestrum are points of differential diagnosis from cystic osteitis.

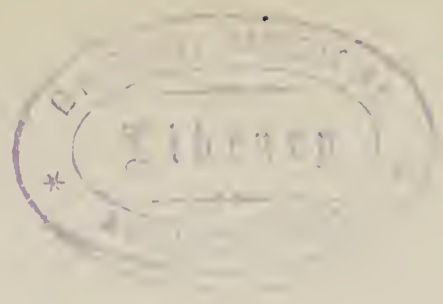


FIG. 15



FIG. 16

FIGS. 15 and 16.—Marked periosteal proliferation of the middle third of the shaft of the left femur. Blood and spinal fluid Wassermann twice negative. Disseminated tuberculosis found at autopsy. No pathological sections were cut from the involved portion of the femur and the diagnosis of tuberculosis of the shaft thus had no absolute verification.



the diagnosis will sometimes be impossible and can only be made, after operation, by other means than the roentgen-ray.

Generalized fibrocystic osteitis should not be so difficult to differentiate from osteomyelitis, but presents more difficulty in relation to syphilis.

CASE REPORT. Because of the rare occurrence of cases of tuberculosis of the shaft of the bone, the following case is reported in detail. Unfortunately, no sections were taken from the portions of the left femur and the left humerus, which were shown to be involved on the roentgen examinations, and so no histological diagnosis of tuberculosis of bone was made. The fact that the left femur seemed to be the part first affected clinically, the negative serological reactions for syphilis, and the widespread occurrence of proven tuberculosis in other parts of the body at autopsy would seem to indicate that this was a case of tuberculous infection of the shaft of the bone.

A. H., aged three years, male, colored, admitted to the service of Dr. Alfred Hand, Children's Hospital, June 21, 1923.

Past History. Tonsillitis, January, 1923; measles, mild, November, 1923; shingles with fever, March, 1923.

Chief Complaint. Inability to walk, with pain in the left leg, especially in the region of the knee.

History of Present Illness. About two weeks before admission began to complain of pain in the left knee. This pain was worse during the morning and disappeared toward midday and evening.

He soon began to limp on the left leg and the pain became so severe that he grasped furniture to support himself in walking, putting almost all of his weight on the right leg. He was brought to the Out-Patient Dispensary, June, 13, 1923, and his mother was advised to put him to bed for a week. During this time the pain became steadily worse. He now does not move the leg unless absolutely necessary. Movement of the right causes him no pain. Appetite is excellent, no vomiting. He has always had a slight cough.

Physical Examination on Admission. Lungs show no impairment of resonance. No adventitious sounds heard. Breath sounds are vesicular. Inguinal glands are palpable.

Extremities. The left femur is of slightly greater circumference than the right (0.5 cm.), when measured in the middle of the thigh. No limitation of motion in any of the joints. Tenderness elicited

on movement of the left hip-joint. No shortening in either leg. Left leg is held in moderate abduction and flexion at the hip with moderate flexion of the knee-joint.

Reflexes. Babinski sign positive, no Kernig's sign, ankle clonus distinct. Rigidity of the neck present, no tache elicited.

Laboratory Findings. June 21, 1923: von Pirquet moderately positive, beginning in twenty-four hours, wheal and erythemia present. Control negative. June 21: spinal-fluid Wassermann, negative. June 28: blood Wassermann, negative. July 6: spinal fluid, Wassermann, negative. September 21: blood Wassermann, negative. October 6: spinal-fluid, colloidal gold test: 0001123554 meningitic curve.

Roentgen Examinations. June 21, 1923: marked periosteal proliferation, with slight cortical thickening of the left femur in the middle third of the shaft and of the left humerus, in the lower third especially along the inner border. Very suggestive of syphilis (Figs. 15 and 16). July 19: examination of the lungs shows an increase in hilum and mediastinal density, probably due to mediastinal and hilum tuberculosis. October 19: the examination of the dorsal and lumbar spine shows no abnormality. No evidence of tuberculous arthritis.

November 11, 1923: Summary of Progress of Case: Since onset, six months ago, the patient has had low irregular fever, 100° F. The pain complained of in the left leg soon subsided. Gastro-enteritis with bloody stools occurred one month after admission, but this also subsided. There has been gradual loss of weight, increasing asthenia and atrophy of the muscles. The patient is mentally below par. Slight exophthalmos of the left eye is now present, with a little increased intraocular tension. The cervical glands are palpable. Examination of the lungs shows nothing striking except perhaps slight dulness and harsh breath-sounds over the left upper lung posteriorly. Liver and spleen are just palpable. The ankle-jerks are hyperactive and equal. Bilateral ankle, clonus is present. Babinski, Brudzinski and Kernig's signs are not present.

Clinical Impression. 1. Syphilis, in spite of negative serological examinations. 2. Disseminated tuberculosis.

December 2: absent knee-jerks; pupils are dilated and unequal. Negative.

December 2: spinal fluid showed many tubercle bacilli, culture for bacteria.

December 5: in the interval since November 18, the child has

become progressively worse; asthenia is more marked, with increasing loss of weight. Patient died at 8.50 P.M.

AUTOPSY FINDINGS. Pathological Diagnosis: Tuberculous meningitis; tuberculosis involving the mesenteric glands, spleen, the third to the sixth dorsal vertebra inclusive, the floor of the middle fossa and anterior fossa of the skull, and the ethmoid sinuses. Small tuberculoma of the dura at the foramen magnum.

Left pleura: Normally smooth, moist and glistening. Behind the parietal pleura is a collection of soft yellow and creamy pus.

Right pleura: Contains about 100 cc. of dark thin fluid. This apparently comes from an opening in the esophagus. There is a collection of pus along the spinal column back of the parietal pleura at the level of the fifth and sixth dorsal vertebræ.

Small intestine shows tubercles in several small areas.

Large intestine shows a few tubercles.

Lymphatics: There is considerable enlargement of the mesenteric glands, which are yellow, soft and cheesy in appearance; some are enlarged to a diameter of 3 cm.

Brain and Skull: There are masses of pus over a large area, which has eroded the surface of the bone in the floor of the middle fossa, left side, and the middle part of the anterior fossa extending down into the ethmoid sinuses. The sella turcica is eaten away and is quite deep. Erosion over the highest portions of each parietal bone is present. There is an exudate of fibrinous character over the surface of the entire brain, with a profuse exudate between the pia and the arachnoid, which covers also the pons and the medulla. On the right anterior edge of the foramen magnum is attached a small irregular mass, 1.5×0.5 cm.

Spinal Cord and Membranes: The cord itself is normal. Between the dura and the vertebræ, from the second to the tenth dorsal vertebra, is much cheesy, pussy material. Between many of the vertebral borders in the vertebral disks are sinuses filled with pus connecting with the pus found in the left pleural cavity. The surfaces of the third to the sixth dorsal vertebræ are quite extensively eroded. The cervical and lumbar vertebræ show no involvement. Bacteriological smears from the pus in the left pleural cavity, skull, meninges and mesenteric glands show an occasional tubercle bacillus.

Histological Sections. Spleen: certain areas show typical tubercles composed of a necrotic center surrounded by small round cells, endothelioid cells and typical giant cells.

Small Intestine: Shows desquamation, also typical tubercles between the mucous and muscular coats.

Mesenteric Glands: The greater part of the sectioned gland is degenerated and consists of necrotic, granular, poorly staining material. The border surrounding shows numerous typical tubercle bacilli.

Esophagus: The site of rupture shows no evidence of an inflammatory condition. The tissue in this area shows only much degenerated poorly staining material with cells made out only more or less distinctly. No evidence found to show that this was an antemortem or postmortem rupture, most probably however it was the latter.

REFERENCES

1. Lovett, R. W., and Wolbach, S. B.: Roentgenographic Appearance, Diagnosis and Pathology of Some Obscure Cases of Bone Lesions, *Surg., Gynec. and Obst.*, 1920, xxxi, 111.
2. Ashhurst, A. P. C., Bromer, R. S. and White, C. Y.: Cystic Disease of bones, *Arch. Surg.*, 1923, vi, 661-730.
3. Brown, C. L. and Stiefel, D. M.: Case of Multiple Bone Lesions of Atypical Roentgenographic Appearance with Pathologic Findings, *Jour. Bone and Joint Surg.*, 1924, vi, 550-563.
4. Allison, N. and Fisher, R. F.: Experimental Bone Tuberculosis, *Am. Jour. Orthop. Surg.*, 1916, xiv, 631-640.
5. Phemister, D. B.: Changes in Articular Surfaces in Tuberculous and in Pyogenic Infections of Joints, *Am. Jour. Roentgenol. and Rad. Therapy*, 1924, xii, 1-14.
6. Codman, E. A.: *Surgery, Its Principles and Practice*, W. W. Keen, Vol. v, p. 1158, W. B. Saunders Company, Philadelphia, 1909.
7. Wilhelm, S. F.: Osteitis Fibrosa and the Hyperostotic Form of Bone Syphilis, *Surg., Gynec. and Obst.*, 1925, xli, 624-639.
8. Baetjer, F. H. and Waters, C. A.: *Injuries and Diseases of the Bones and Joints*, Paul B. Hoeber, Inc., New York, 1921.
9. Bloodgood, J. C.: The Diagnosis and Treatment of Benign and Malignant Tumors of Bone, *Jour. Radiol.*, 1920, i, 147.
10. Ewing, James: *Neoplastic Diseases*, W. B. Saunders Company, Philadelphia, 1922.
11. Codman, E. A.: The Nomenclature Used by the Registry of Bone Sarcoma, *Am. Jour. Roentgenol. and Rad. Therapy*, 1925, xiii, 105-126.
12. Wimberger, H.: Zur Diagnose des Sauglingsskorbut., *Ztschr. f. Kinderh.*, 1923, xxxvi, 279-285.
13. Pelkan, K. F.: The Roentgenogram in Early Scurvy, *Am. Jour. Dis. Child.*, 1925, xxx, 174-188.
14. Lovett, R. W.: The Roentgenographic Appearance in Rickets, *Jour. Am. Med. Assn.*, 1915, lxv, 2062-2067. (Also in Transactions of the Section on Orthopedic Surgery, American Medical Association, June 22, 1915.)

MULTIPLE GIANT-CELL TUMORS. REPORT OF A CASE AND REVIEW OF THE LITERATURE*

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GIANT-CELL tumors have held a very prominent place in American studies and discussions of bone pathology since Bloodgood, in 1910, called attention to the benign character of this condition and in 1912 recommended the name which now is in common use. Single giant-cell tumors are not uncommon. The literature contains reports of several hundred cases and there are undoubtedly many more that have not been reported. Multiple giant-cell tumors, however, are sufficiently rare to warrant our reporting this case with a résumé of the previous cases which we have found in American and foreign literature.

CASE.—J. M., aged twenty-two years, a well developed, well nourished, but rather anemic white male, was admitted to the Episcopal Hospital, service of Dr. Alexander, on October 20, 1926, with a fracture of both femurs in the middle third and the right humerus at the junction of the upper and middle third.

Family History. Father, mother, four brothers and one sister living and well. Grandmother died of cancer. Family history otherwise negative.

Personal History. Measles, chickenpox and tonsillitis in childhood. Operation for varicocele at the age of fourteen years. Operation for a ruptured right ligamentum patellæ, January, 1925 (*x*-rays of the right knee taken at that time showed beginning bone changes). Injury to the right arm, two weeks before admission, showed no fracture in *x*-ray, but a pathological bone condition

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suggestive of chronic cystic osteitis of the shaft of the right lower humerus and ulna; punched-out areas were seen in the lower end of the humerus and periosteal proliferation along the border of the ulna, which in certain places had a lacework appearance.

The patient's habits are good. He has considered himself in good health, but gives a history of rheumatism in the knees for the past four months, with sharp, deep pains in the legs, which were worse in bad weather. He has lost twenty pounds of weight in the last five weeks. No history of venereal infection.

Present Illness. Patient slipped and fell in the bathtub and broke both femurs and the right humerus. He was brought immediately to the hospital. Buck's extension and Volkmann's sliding splints were applied to both legs and the right arm was dressed with a shoulder-cap and right-angle splint. This was changed after several days to a weight-extension dressing.

Physical Examination. Patient is a well-nourished white male, about 5 feet 10 inches in height and weighing about 180 pounds, appearing rather anemic.

Head. Is negative, except that the teeth are in poor condition and pyorrhea is present. There is a hoarseness of the voice, which was later found to be due to paralysis of the left true vocal cord, which had come on following an anesthetic eighteen months previously. There is a small tumorous swelling in the right mandible near its angle, the result of an injury while boxing.

Chest. Heart and lungs are negative. No nodules are found on the ribs.

Abdomen. Negative.

Extremities. Show fractures of both femurs and the right humerus, with displacement, pain swelling and crepitus. There is a scar over the right knee from previous operation. There is swelling of the lower end of the left radius.

Progress Notes. October 20, 1926: *x-ray* examination. Besides the fractures, all bones were involved in a pathological condition and was interpreted as follows:

"There is a generalized fibrocystic osteitis with an atypical appearance in different regions, which in the distal end of the radius and the lower extremity of the left femur has advanced to that of a giant-cell tumor; in the region of the elbows it has the appearance of a chronic osteomyelitis; in the right femur the appearance suggests strongly the possibility that the process has undergone a sarcomatous degeneration."

November 8, 1926: Biopsy of the tumor of the left radius. Preoperative diagnosis, giant-cell tumor. Under nitrous oxide and

oxygen anesthesia an incision 4 cm. long was made on the dorsal aspect, over the tumor, of the lower end of the left radius. Cultures were made from the skin, subcutaneous tissues, fascia, periosteum and medullary cavity. When the periosteum had been shoved aside with a small separator, a piece of bone sufficient for study was quite easily removed. It was shell-like, about 1 mm. in thickness and the curette easily broke through into the medullary cavity. The contents of the cavity was a soft, red, bloody spleen-like pulp. Some of this material was also taken for study. There was a slight bloody ooze, but the incision was closed completely with interrupted silkworm-gut suture. The wound healed by first intention within ten days. Pathological report of biopsy was giant-cell tumor, and the cultures taken were negative for organisms.

November 23, 1926: X-ray of the lower end of both tibias and fibulas and of the bones of the feet and ankles showed the same moth-eaten areas as elsewhere. The pelvis showed mottling and there was a pathological fracture of the femur of the left leg, not discovered clinically. X-ray of the chest showed the lungs not infiltrated with metastatic neoplasm, but the eleventh left rib showed a tumor at its angle.

December 9, 1926: Biopsy of the left ulna. Preoperative diagnosis, chronic osteomyelitis. Incision was made over the upper part of the ulna, so that the bone was exposed about 5 cm. below the tip of the olecranon. Cultures from the skin, fascia, periosteum and bone were taken. The bone was found to be roughened and not as hard and compact as normal bone. No medullary substance was exposed. A small piece of bone was removed and the incision closed. The wound healed by first intention. Pathological report was giant-cell tumor and the cultures were sterile.

Biopsy of the tumor at the lower end of the right femur revealed a condition similar to that of the left radius. The bone tissue was shell-like, 1 or 2 mm. thick, and the marrow a spleen-like pulp; there was a great deal of hemorrhage. Bone and marrow tissue were removed for study and cultures were taken as in previous operation. A rubber-tissue drain was inserted and the incision closed with interrupted sutures. The wound healed by first intention except at the site of the drain and this healed by rapid granulation. Preoperative diagnosis was sarcoma. Laboratory diagnosis was giant-cell tumor and again the cultures showed no growth.

December 13, 1926: Areas of right radius, left ulna and right

femur, from which biopsies were taken, have a more extensive moth-eaten appearance, as shown by *x*-ray.

December 31, 1926: Clinically there is only fibrous union of the fractures. All weight extensions have been removed (seventy-two days after admission).

January 28, 1927: By *x*-ray the fracture of the right humerus showed excellent callous formation (100 days after fracture). "There should be some bony union. There also seems to be some calcium deposition within the bone. There is practically no change in appearance of the left forearm. There is slight callous formation about the fracture of the left femur, but very little if any calcium deposition."

January 31, 1927: Patient is up in a wheel-chair. He can use his right arm to feed himself. He has a good appetite and is clinically improved. There is a false joint at the site of fracture. There is nonunion and nonuse of the femurs.

Laboratory Data. Urine negative for Bence-Jones albumin on three occasions; a trace of albumin with an occasional granular cast. Blood urea nitrogen and blood-sugar, normal. Icterus index, 11.2. Van den Berg, negative. Hemoglobin, 50 per cent; red blood cells 2,830,000; white blood cells, 20,600; polymorphonuclears, 82 per cent; transitionals, 1; lymphocytes, 15 per cent; eosinophiles, 2 per cent; anisocytosis and poikilocytosis present; blood calcium, 15.4 mg. per 100 cc.; blood phosphorus, 2.1 mg. per 100 cc. Spinal and blood Wassermann, negative. Spinal colloidal gold, negative. Spinal fluid cells, 2 per c.mm.

CASES IN AMERICAN LITERATURE

CASE I.—CRILE AND HALL. A young unmarried woman, aged twenty-two years, *x*-ray pictures of the skeleton showed numerous bones involved in a pathologic process. Operation was performed on a lesion in the right tibia; gross and microscopic study was made of the contents and a diagnosis of multiple giant-cell tumor was made. The etiologic factor was not ascertained. This history states that the patient's mother had syphilis and her father diabetes. The patient had no evidence of acquired lues and also failed to improve on antiluetic treatment. However, it was noted that the exploratory wound gave no evidence of healing until the patient was put on iodide therapy; prompt healing of the wound then resulted. Ten years later (1915), when the case was seen by Dr. Hirsch in the Bellevue Hospital, there were numerous masses



FIG. 5.—Left radius and ulna showing a giant-cell tumor.



FIG. 6.—Left ulna showing the area from which biopsy was taken.

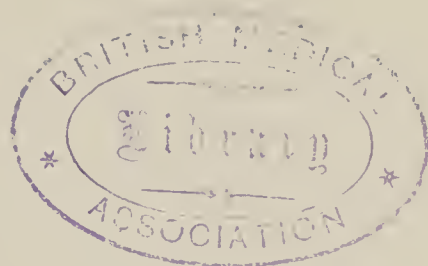


FIG. 7.—Both hands: showing a giant-cell tumor of the distal end of third metacarpal, right hand, and showing also the typical moth-eaten appearance of bones of the hands.



FIG. 8.—Right knee showing evidence of a giant-cell tumor at upper end of the tibia.

apparently attached to all the bones. X-ray revealed multiple multilocular cystic tumors, which were diagnosed multiple giant-cell tumors.

CASE II.—HARTUNG AND KANAVAL. Male, aged thirty-four years, a carpenter, admitted to the hospital with an ununited fracture of the left femur sustained six months previously. Patient walked with a cane and crutch. A diagnosis of bone cyst was made. Four years later he had involvement of both clavicles, several ribs, right ulna, tibia and fibula, right humerus and femur, left tibia and one metatarsal, and an old fracture of the left hip. Three years later the case was presented to the Chicago Surgical Society. Several cysts had been scraped out and revealed a pathologic picture of giant-cell tumor.

CASE III.—HAUSSLING AND MARTLAND. A married woman, aged twenty-five years, has had four normal deliveries and one miscarriage in which there was much hemorrhage and following which she had weakness, dyspnea, palpitation on exertion. She fell and broke a femur, 6 inches above the knee, in October, 1914. There was union without deformity. May, 1915, she had a full-term labor. In June, 1915, she was admitted to the hospital because of weakness and dyspnea. Examination of the heart showed a loud systolic murmur and the lungs with signs of early tuberculosis in the left upper lobe. Palpable tumors were found in the right orbit, both clavicles, left tibia and seventh right rib, and further, by x-ray, in the right femur, right and left fibulas, right humerus and in the pelvis. Biopsy of the growth on the left tibia showed a characteristic giant-cell tumor. Curettage of several tumors was later done. Incisions united by primary union. The tumors recurred and later others were also found elsewhere.

CASE IV.—BARRIE. White male, aged fifty years, married, youngest child eighteen years of age. Wife had no miscarriages. Denies venereal infection. Had sciatic rheumatism fifteen years previously, attacks of weakness of legs and feet eight years ago, and again seven years ago. All teeth extracted six years previously. Diagnosed amyotrophic lateral sclerosis five years ago. While on his way to the hospital for this admission he had an accident and fractured the eighth, ninth and tenth ribs. X-ray showed areas of osteolysis in the long bones of the lower extremities, the ribs on the right side, the fractures being through the pathological areas.

There was a mass on the right tibia the size of a hen's egg. Diagnosis of multiple gumma was made and treatment given without result. Exploratory operation of the tumor on the left tibia revealed giant-cell tumor.

CASES IN FOREIGN LITERATURE

CASE I.—HIRSCHSPRUNG. Female, aged thirty-five years, fell and fractured the left hip. Had union with shortening; also had a periosteal swelling of the left shin bone. Four years later she was admitted and treated for rheumatism and periostitis, and died of marasmus. Postmortem showed no changes in internal organs. Left humerus had a healed fracture at the anatomical neck, the left tibia a healed fracture in its middle, and the left femur a healed fracture at its neck. The bones at the site of fractures were soft and cystic and in the tibia at the site of fracture was a small giant-cell sarcoma.

CASE II.—SCHOENENBERGER. Female, aged thirty-three years; no previous illnesses. During her third pregnancy developed pain in the back and limbs, which persisted after labor and was treated as a chronic articular rheumatism. She was admitted to the hospital because of this pain and acute joint-pain followed by swelling of the bones. She had multiple fractures, due to slight trauma. Autopsy later showed fracture of the right and left humerus, right and left femurs, lordosis of the lower thoracic vertebræ, fracture of numerous ribs, cystlike tumors of the left tibia and right tibia and fibula and humerus, both femurs, pelvis, etc., which proved microscopically to be giant-cell sarcoma.

CASE III.—SCHLANGE. Male, aged eighteen years, had a fracture in the middle of the femur five years previously. After healing, there were pain and deformity of the leg. Operation revealed a cyst 3 cm. long with serous fluid, and a second cyst the size of a walnut, extending into the greater trochanter. Microscopic examination showed the bases of the tumor masses to be cellular connective tissue with delicate bone formation, and in the region of the area of softening numerous giant-cells.

CASE IV.—REHN. Female, aged twenty-three years; admitted because of pain in the right hip, with visible swelling. Discharged after two and one-half months slightly improved. Readmitted ten months after onset, with pain and a rapidly growing tumor at

the distal end of the right ulna. It was removed at operation. Examination revealed a gray, red, friable mass and microscopically giant-cell sarcoma. Two months later the shaft of the right femur, right ileum and right humerus were affected. Biopsy of the right ileum tumor revealed a giant-cell sarcoma. Several months later the right lower leg, eighth and ninth ribs, left sacroiliac joint and left tibia were all involved. Operation on the sacroiliac joint, because of pain, showed giant-cell sarcoma also. One year later, tumors had increased in size and number and there was a spontaneous fracture of both femurs, which healed. There was progressive deformity due to fractures and softening of the bones. Patient died four years later of anasarca.

CASE V.—HABERER. Boy, aged ten years, well until three years ago, and has developed irregular, increasing, painless swellings of the right side of the head and face. There was a five-year-old slight traumatic fracture of the right femur in the middle third which had healed with deformity, and three years later slight trauma had increased the deformity. X-ray showed lesions in the right parietal region, lower jaw, right femur, left trochanter, left coxa vara. Exploratory operation showed that, after cutting through the thin cortex, multilocular hemorrhagic cavities filled with soft red-brown sarcomatous masses were found. Microscopically it showed widely disseminated giant-cells and spicules of bone. Diagnosis: Giant-cell sarcoma. Progress was benign.

CASE VI.—HART. Female, aged seventy-eight years, well until sixty-eight years of age, and lived in an old folks' home, had a spontaneous fracture of the femur while lying in bed. She died of hypostatic pneumonia and suppurating bronchitis. At post-mortem, skeletal tumors were found in both femurs, both tibias, pelvis, ribs, right humerus, elbow, radius, eighth and ninth thoracic vertebra, all due to giant-cell sarcoma and cysts.

CASE VII.—GUENTHER. Reported a case of Fischer's. A carpenter, aged forty-six years, came to autopsy with a diagnosis of osteomalacia. Anatomical diagnosis was multiple tumors, myelomatous and myelosarcomatous, throughout the bony system, with fractures of both femurs, the left humerus, and a tumor of the right parathyroid. Microscopically the bone tumors showed the picture of giant-cell tumor of the epulis type.

He also quotes Schmoil as reporting four cases of osteomalacia with parathyroid changes, and Mollineau's case of osteitis fibrosa

with multiple giant-cell sarcoma in which three of the parathyroid bodies showed changes. He also collected two other cases to show the relation between malacia of bone and parathyroid bodies.

CASES OF FIBROCYSTIC OSTEITIS WITH ASSOCIATED MULTIPLE GIANT-CELL TUMORS

Morton has carefully studied and analyzed many cases of fibrocystic osteitis with giant-cell sarcoma from which we briefly quote the following cases:

WERNDORFF. Male, aged nine years, with tumors in the right femur and right tibia, duration from earliest childhood, causing deformity of the right leg. Resection of the tumor of the right femur proved to be giant-cell sarcoma.

BUTLIN. Male, aged fifty years, who since he has been forty-three years of age has had tumorous masses growing on the jaw and on the sixth right rib. At autopsy these were found to be giant-cell tumors.

Female, aged forty years, had tumorous formations on the upper and lower jaw, duration ten years, causing swelling, and when removed and studied were found to be giant-cell tumors.

DAVIDSOHN. Male, aged fifty-eight years, with tumors of the tibia, patella and femurs, discovered five years previously. Came to autopsy and showed giant-cell tumors.

GAUGELE. Female, aged thirty-six years, who had shown symptoms since twenty-eight years of age, the tibia, humerus and ulna being involved. The symptoms were fractures, swellings, deformity, anemia and emaciation. Pathological report indicated giant-cell sarcoma.

VON RECKLINGHAUSEN, 1891, CASE VII.—Female, aged forty years, with tumor of the left ilium, upper and lower jaws, radius, tibia, ribs, fibula and femur, and who had been treated for pain, fractures and deformity, died of marasmus. The pathological report included giant-cell sarcoma.

LOTSCH. Reported a case, aged fifty-seven years, who dated the onset of his condition ten years previously, who showed tumors

of both tibias. Exploratory operation was done which showed giant-cell tumors.

MONCKEBERG. Female, aged fifty-five years, who since thirty-nine years of age has had a tumor of the jaw and another tumor of the ninth rib. The tumor of the jaw was extirpated three times and it showed a giant-cell tumor.

CASES SUBMITTED TO THE SARCOMA REGISTRY

From a recent communication from Dr. B. C. Crowell, we add the following cases which have been submitted to but not definitely decided upon by the Committee on Bone Sarcoma:

CASE 70.—DR. R. E. FORT, Nashville, Tenn. Date of onset about November 12, 1912. A boy, aged eleven years, with a tumor of the first rib. July 23, 1913, excision of first rib, clavicle and part of sternum. Postoperative Coley toxins. Registrar's classification—Giant-cell tumor. Late note, May, 1923—well. Published in *Surgery, Gyn., and Obst.*, June, 1914, pp. 696-698. September, 1924—well. May 1, 1925—well.

CASE 110.—DR. J. C. BLOODGOOD, Baltimore, Md. Date of onset about 1917. A man, aged twenty-seven years, with a tumor of the upper end of the tibia and femur. July, 1920, exploratory incision by family doctor. Sinuses followed. November 27, 1920, amputation of femur by Dr. Carr. Registrar's classification—giant-cell tumor. Last note March, 1921—well.

NOTE.—A recent communication from Dr. Bloodgood states that he has three cases of multiple giant-cell tumors which he has not published.

CASE 167.—DR. JAMES EWING, New York, N. Y. Date of onset January, 1918. A boy, aged ten years, with a large tumor of the pubis, ischium and acetabulum. Pathologic fracture. Very clearly a giant-cell tumor from x-ray. No incision. Treatment: Fixation and radium. Registrar's classification—giant-cell tumor (x-ray diagnosis only). Last note, June, 1923—well, good function. July 12, 1924, no change. July 9, 1925—well. October, 1926, no further report.

CASE 212.—DR. DAVID CHEEVER, for the Peter Bent Brigham Hospital Clinic. Date of onset April, 1920. A man, aged twenty-

four years, with a tumor of the lower end of the left tibia and the fibula. Previous operations, about July, 1920, diagnosis was made of giant-cell sarcoma; recurrence and second operation March, 1921, followed by radium and *x*-ray treatments. Incision has never healed, continued lameness and soreness, although patient was able to work. Admitted to Peter Bent Brigham Hospital, April 20, 1922. Examination showed nearly complete destruction of lower end of tibia, marked involvement of fibula and invasion of ankle-joint. Patient developed a severe pyogenic infection from the unhealed wound from which tumor tissue was sprouting and had to have multiple incisions of leg and thigh. Amputation of the lower leg was done July 13, 1922. Patient discharged with stump well healed on July 28, 1922. *X*-ray of rest of skeleton and lungs negative. Pathological examination—giant-cell tumor. Registrar's classification—giant-cell tumor. Last note, April 7, 1924, well, wearing artificial leg. July 23, 1926, no further report.

CASE 590.—DR. G. E. PFAHLER, Philadelphia, Pa. Date of onset, 1919. A man, aged forty-two years, with a large tumor involving upper end of the femur, pubes, acetabulum and ischium. Case diagnosed as osteosarcoma at Samaritan Hospital, May, 1922. October 9, 1922, admitted to Medico-Chirurgical Hospital. Pain first noticed in the knee three years before admission and a little later pain in the left hip. Eleven months before admission noticed a lump in the left groin. On admission, leg flexed and unable to step on foot. Treated by *x*-ray from October 9, 1922, to October 16, 1923. Marked improvement, and in two months was able to leave the hospital. Registrar's classification—Benign giant-cell tumor. Last note, October, 1924, has been using the leg and working for the past year and a half. To have further radiation because of arrest of calcification in tumor. February 10, 1925, further *x*-ray studies showed that the head of the femur is destroyed. Having increased pain. No increase in calcification. Hip-joint ankylosed.

DISCUSSION. Giant-cell tumor, giant-cell sarcoma, hemorrhagic osseous dystrophia, myeloid sarcoma, myeloma, osteitis fibrosa with giant-cells, chronic hemorrhagic osteomyelitis and "brown tumors" are all terms used with reference to a similar bone condition. The subject was much studied between 1840 and 1860. Lebert, in 1845, was probably the first to recognize the condition. It was later described by Paget in 1854, and later by Nélaton in 1860 in an elaborate monograph.

Nélaton emphasized the proliferation of giant-cells, myelopacques, as the essential factor in the process. He insisted that the giant-cells must predominate in the tissue and not be present merely in small numbers, since such cells were occasionally seen in other tumors. He recognized several other anatomical varieties, depending on location, conformation, structure and stage of evolution of the tumor. The age of incidence was mainly between fifteen and twenty-five years. Without the aid of the microscope diagnosis was usually impossible. Regarding prognosis, Nélaton was quite specific, saying that every tumor composed essentially of giant-cells should be regarded as benign. He also advocated cauterization with zinc chloride following curettage, since the tumor would generally recur if any fragment was left.

Virchow (1864) is often quoted as emphasizing the malignant behavior of certain myeloid sarcomas, but was unable to demonstrate that any of his malignant cases had not been such from the beginning.

Gross, in 1879, described in detail the features of giant-cell sarcoma, analyzing 70 cases from various sources and emphasizing their benign character.

In America, the facts established regarding giant-cell tumors seem to have been largely disregarded for many years and most of the tumors were subject to radical operation, until Bloodgood, in 1910, called attention to the benign character of the disease.

Giant-cell tumor is a specific tumor, believed to take origin from the fibrous-tissue framework of the bone, whether periosteum or endosteum, and characterized by the invariable presence of osteoclast-like giant-cells in large numbers. It has at various times been considered the result of bone destruction due to spirochete, tuberculosis, infectious bacteria and parasites, trauma, malnutrition and metabolic change. While it must be recognized that the solitary local process and the multiple systemic lesions give exactly similar gross and microscopic pathological findings, it is also quite clear that the etiological factors bringing about these apparently identical conditions are varied. In most cases the cause of solitary lesions is trauma. Von Recklinghausen regarded the systemic

multiple lesions as different forms of malacia, Guenther, more recently, believes it is due to a lack in the balance of bone chemistry associated with parathyroid disturbance.

Giant-cell tumor classification has created much discussion. One school considers it neoplastic and another inflammatory, while a third states that it is a borderline lesion between these two. It is generally considered, however, as resulting from some chronic irritation, which may follow a metabolic disorder. The lack of calcium deposition weakens the bony structure; local injury, the stress and strain of motion and work, organisms, etc., are all irritants.

The inflammatory proliferation of tissue is then essentially a regenerative process which has for its aim the compensation of the lesion produced by the cause of inflammation. Under special conditions this leads to a hyperplastic proliferation of connective tissue, frustrates its own aim and causes new damage. This is particularly the case when, as a result of the inflammation in the organism, there is kept up a permanent condition of inflammation. The bone tissue thus replaced by cellular tissue, softens and produces multiple bone cysts lined with fibrous tissue and filled with clear fluid, fibrocystic osteitis, or within the fibrous tissue lining the giant-cell tumor develops.

The giant-cell tumor usually arises in the interior of the shaft of long bones near the epiphyses. It is of slow growth, does not produce metastasis or cachexia, expands the bone abruptly, and in the *x*-ray appears trabeculated. The bone may be so thin as to crackle, and when cut is quite soft, vascular and resembles splenic tissue, but having a firmer opaque texture on the surface and central softer, cystic, or hemorrhagic areas. There is seldom any tendency toward invasion of the soft parts. Microscopically the framework has much the appearance of granulation tissue with hemorrhagic areas and abundance of large giant-cells containing many small oval nuclei.

According to Mallory, there are two types of giant-cells, a tumor giant-cell and a foreign body giant-cell. The former are large, clear, bladder-like cells, with distinct outline but staining faintly, within which are multiple nuclei, or a large multilobulated nucleus with mitotic figures, which stain deeply

and are situated in the center of the cell. They are usually not important features of the microscopic picture, but may be numerous and conspicuous. They are true tumor cells resulting from multiple mitosis and signify rapid growth. The second type are as a rule smaller, their cytoplasm fairly abundant, sharply defined and staining deeply with acid dyes. The nuclei are smaller, uniform, more numerous, without mitosis, and are often in clusters near the periphery of the cell. They resemble osteoclasts and are merely a reaction to the presence of foreign bodies and are due to the fusion of endothelial leukocytes.

Von Hansmann has classified giant-cells briefly as follows:

1. Foreign body giant-cells of endothelial or leukocytic origin.
2. Parenchymatous giant-cells, tumor-cells proper, due to irregular mitosis and lack of cell division.
3. Myelopaxes, present normally in red bone-marrow and characteristic constituents of myelomata.

The giant-cells of our own giant-cell tumor would be in Mallory's class two, and von Hansmann's class one.

Giant-cell tumors may be present an indefinite time without giving rise to any symptoms. Often the first indication of their presence is the occurrence of pathological fractures. In our case, after reviewing the films, x-ray shows beginning bone changes eighteen months before the patient was brought to the hospital with symptoms; also, we have done biopsy on areas absolutely symptom-free and found early giant-cell tumor formation. However, according to the location of the tumors, they may cause pain due to expansion and pressure on the soft parts.

Diagnosis is made by biopsy and microscopic examination, or may in a few cases be made by roentgenogram.

The treatment of single or multiple giant-cell tumors, when few in number, consists of thorough curettage and the application of pure carbolic acid, followed by the use of alcohol, or perhaps, better still, 20 per cent zinc chloride. The cavity is then kept clean with Dakin's solution until healed. Coley, in 1924, advocated the additional use of mixed toxins of erysipelas and bacillus prodigiosus, given systematically for a period of three or four months, and if available, one massive dose of

radium, made over the tumor after the danger of infection is over or when the sinus has entirely healed. X-ray treatment is said to give great benefit, if not a cure.

CONCLUSIONS. 1. Giant-cell tumors, especially multiple tumors, are being found more prevalent than previously, due to the x-ray.

2. No cases of multiple giant-cell tumors have been accepted as true entities by the Committee on Bone Sarcoma. (Dr. Codman, in a recent communication, states that he is skeptical about the existence of the condition.)

3. We report this case without knowing what previous pathological condition existed at the areas biopsied, nor what change may take place in the course of a few years, should the patient live, but we have selected areas which should show different stages and we have found them to be multiple giant-cell tumors.

4. We have attempted to collect the cases from the literature in which multiple giant-cell tumors were believed to exist either alone or in conjunction with other bone changes.

REFERENCES

- Adami: Principles of Pathology, vol. i, 1910.
 Ashhurst: Surgery, Principles and Practice, 1920.
 Barrie: Annals of Surgery, 1920, lxxi, 581.
 Bloodgood: Annals of Surgery, 1910, lii, 145; 1912, lvi, 210; 1919, lxix, 345.
 Progressive Medicine, December, 1918. Surg., Gynec. and Obst., 1924, xxxvii, 784.
 Coley: Inter. Jour. Surg., 1924, xxxvii, 81-89.
 Connell: Surg., Gynec. and Obst., 1915, xx, 427.
 Crile and Hill: Surg., Gynec. and Obst., 1906, iii, 57.
 Elmslie: Brit. Jour. of Surg., 1914, ii, 17.
 Ewing: Neoplastic Diseases, Second Edition.
 Guenther: Frankfurt. Ztschr. f. Path., 1922, xxvii, 298.
 Haberer: Archives of klin. Chir., 1907, lxxxii, 873.
 Hart: Ziegler's Beitr. z. Path. Anat., vol. xxxvi, p. 353.
 Hartung: Am. Jour. Roent., 1914, i, 201-208.
 Hirschberg: Ziegler's Beitr. z. Path. Anat., 1889, vi, 511.
 Kanavel: Surg., Gynec. and Obst., 1915, xx, 745.
 Mallary: Principles of Path. Hist., 1914.
 Martland: Proc. N. Y. Path. Soc., 1915, xv, 119.
 Martland and Haussling: Annals of Surgery, 1916, lxiii, 454.
 Morton: Archives of Surgery, 1922, iv, 534.
 Myerding: Jour. Am. Med. Assn., 1924, lxxxiii, 1323.
 Platon: Annals of Surgery, 1918, lxvii, 312.
 Rehn: Archives of klin. Chir., 1904, lxxiv, 426.
 Schlange: Archives of klin. Chir., 1902, lxviii, 992.
 Schoenenberger: Virchow's Arch., 1901, clxv, 189.
 Stewart: Lancet, Lond., 1922, ii, 1106-1108.
 Stone and Ewing: Archives of Surgery, 1923, vii, 280-296.

THE PROGRESS OF OPHTHALMOLOGY IN THE PAST SEVENTY-FIVE YEARS IN RELATION TO GENERAL MEDICINE

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THE progress of ophthalmology and medicine during the past seventy-five years has been due, in large measure, to the greater accuracy of observation and to the increase in the instrumental armamentarium, which have enabled the physician to know positively the facts that he had previously surmised. As long as a diagnosis was dependent upon assertion, the patient was necessarily guided by the personality of the physician; but when the diagnosis could be proved to the satisfaction of any disinterested person, the position of the theoretical logician or irregular practitioner was rendered more difficult.

The younger generation is no longer almost completely dependent upon apprenticeship in clinics before being shown the intricacy of diagnosis, but is given instruction in post-graduate courses in the use of instruments of precision that permits him to ascertain positive knowledge upon obscure points to assist him in making his deductions. Experience, however, remains essential in its correct interpretation, and the hospital clinics are the source of this important element of instruction.

Ophthalmology is one of the oldest of specialties and was necessarily one of the first in which accuracy was essential. Its progress through centuries was limited; but when the gateway to the interior of the eye was opened by Helmholtz, in 1851, in his historic treatise, *Beschreibung eines Augenspiegels*, new life was injected. The optic nerves, arteries, veins, retina and choroid were visible to anyone that possessed the skill to use his ophthalmoscope.

Loring was the first successfully to add lenses before the opening in the mirror so that ametropic eyes could be examined and the refraction estimated with the ophthalmoscope. Since that time many improvements have been made by different individuals. While the foundation was laid by Helmholtz and Loring, the use of the ophthalmoscope was largely limited to ophthalmologists until the introduction of the electrically lighted ophthalmoscope, which made it possible for any one without much previous experience to see the interior of the eye and to note gross changes. The correct interpretation of these conditions may not be so easy. Nevertheless, the electric ophthalmoscope has opened a way for the general practitioner to evaluate the common and pronounced changes in the eyeground. Due to the ophthalmoscope, the relation between ocular disease or symptoms and the general system has been made more manifest. When a choked disk or optic neuritis is found we look elsewhere than in the eye for the ultimate cause of the poor vision: We examined for brain tumor or other cause of intracranial pressure which can be relieved by decompression of the skull. Chronic interstitial nephritis can cause a similar ocular picture, though usually the nerve head is not so much swollen. Ordinarily, in the early stages of contracted kidney there is retinal edema and hemorrhage, while in the later stages the macular region is occupied by many spokes of small white dots emanating from the macula as the hub. In fractures of the skull and in interference with the venous circulation of the brain, as in sinus thrombosis, unilateral swelling of the nerve head and engorgement of the veins are of much significance in locating the lesion.

Time does not permit any lengthy discussion of the many relationships existing between the body and the window of the soul. We wish to mention only a few and relate some of the improved methods of diagnosis and treatment.

An important feature in medical diagnosis has been the development and understanding of visual fields. The advanced ophthalmologist has made use of this procedure for fifty years or more, but its extended employment in the field of neurology has been of great importance. A brief review of some of the

striking results of the use of perimetry may not be amiss. The recognition of pituitary diseases, for instance, is shown in hemianopic fields for form and color. We have contractions of the field for color or form or both in various optic atrophies due to spinal diseases. The central scotomas of tobacco and alcohol poisoning are shown first as a relative shading in the perception of red and green at the fixation point. This visual-field loss, in later stages, becomes absolute for white and colors, thus causing the greatly reduced vision in these cases.

The cure of headache by the use of properly curved astigmatic lenses to correct corneal astigmatism was largely due to the researches of Philadelphia physicians, especially worthy of mention, being Doctors S. Weir Mitchell and William Thomson. William Zentmayer, the father of one of the most distinguished of our present-day ophthalmologists, made the first astigmatic lenses in Philadelphia upon prescription. In much later years, Dr. George Gould, with his virile writings, attracted the attention of the profession to the cure of other obscure and remote symptoms by careful refraction of the eyes. His statements regarding the efficiency of glasses to cure every headache, appendicitis, tuberculosis, melancholia and a host of other general diseases may be regarded as enthusiastic. It is true, however, that very small errors of refraction and very slight muscle errors cause severe and unexpected headache and distress, which are immediately relieved with proper glasses. The reflected pains are due probably to association fibers, causing local and general nerve exhaustions. A symptomatic wryneck has been produced by palsies of the ocular muscles, causing the patient to assume an improper posture to secure monocular vision; a cure followed proper correction of the condition. The refraction of the eye has made tremendous strides, in the past fifty years, in the realization of the necessity of extreme accuracy in the estimation of astigmatism and its proper angle. For this purpose many instruments of precision have been invented and are of assistance. This apparent simplification has given rise to a new cult, who call themselves "optometrists," who assume the title of doctor and who charge a fee for their

professional service. They supplant the optician, who was satisfied with his profit as a jeweler, on the side, and who would largely limit himself to spherical lenses to correct hypermetropia, myopia or presbyopia. The optometrist has a better education and has discovered a short cut to the medical profession, doing away with unnecessary time and money spent in a scientific college, followed by a medical college and with at least one year of hospital experience. I mention this viewpoint as it is a source of regret that some medical physicians forget their sense of pride in our profession sufficiently to, at least occasionally, refer patients to representatives of this cult. If these mistaken medical brethren would investigate, they would find that the incomplete education has made the optometrist find astigmatism where there is none. He would see that eyewaters, eyesalves and other treatment are prescribed by the optometrist in the shape of ready-made proprietary medications. There is no law against selling yellow oxide of mercury salve or boric acid solution, etc., and the optometrist desires to be considered a doctor. Invasion of the medical ranks by the insufficiently prepared individual is not a new thing in the history of medicine: Seventy-five years ago, in the State of Pennsylvania, outside of Philadelphia, any person could practice medicine without demonstrated previous training and without a license.

In the early days of refraction, atropin or belladonna drops, as they were called, were used in every case, and long enough in the young, to cause a complete paralysis of the iris and ciliary muscle. Much ado was made by enthusiasts when an extra fractional diopter could be added to the refraction under a mydriatic. The patient had to accustom himself to the lenses, often with much discomfort. At the present day, with retinoscopy and a temporarily dilated pupil, an accurate knowledge of the static refraction is obtainable. The amount of spasm remaining in the ciliary muscle will show in the difference between the retinoscopic and test examination. A positive determination is then possible as to the wisdom of further mydriasis and a full correction.

In the matter of external manifestation of disease in the eye, accurate knowledge, made possible by the general advance

of all of the specialties, enables us to diagnose and operate if necessary on conditions that were obscure in early days. The bulging of the eye forward (unilateral exophthalmos), which we have seen rather frequently of late, due to acute ethmoidal sinusitis, is quickly recognized and treated as an acute sinusitis and not as a cellulitis, as formerly. Rapid improvement follows intranasal treatment, though operation is occasionally indicated. In children this affection can rapidly spread to the cavernous sinus with fatal results. An early drainage externally is simple, efficient and practically free from danger. An involvement of the cavernous sinus is indicated by beginning exophthalmos of the opposite eye and is extremely dangerous to life.

The relation between phlyctenular disease and eczema has always been recognized to some extent. In nearly every instance there is a positive von Pirquet test found in these children. This does not always indicate an active tuberculous infection, but may show a latent form of the disease. A proper diet with rest quickly cures the phlyctenular ulcers after the eye is put to rest with atropin solution. The use of old tuberculin in very minute doses has proved of great value in the persistent tuberculous forms.

Iritis is due to a toxic infection of some kind and frequently it is of focal origin, though it was thought formerly to be entirely due to rheumatism or syphilis. In every case of iritis it is now deemed essential to examine tonsils, teeth, sinuses, gall-bladder, genitourinary tract, etc. Neisserian infection of the urethra, causing iritis, can be combated by vaccine injections. In the early days of this hospital, focal infections were never considered and the importance of micro-organisms not realized.

The injection of antitoxins and bacterins is very efficient in certain types of severe infections of the eye. Foreign proteins, such as boiled milk, sometimes are used with success. A knowledge of general medicine and surgery is necessary to produce an eye physician of the modern type, and this close alliance between medicine and all its specialties is being recognized with our increasing knowledge.

We cannot accomplish thoroughness in any branch except

by exclusive practice of this specialty; but our study must keep us in contact with the advanced practice in every other specialty. The medical practitioner should be able, with the aid of the ophthalmoscope, to recognize gross abnormalities such as marked swelling of the optic nerve, hemorrhages of the retina and disease of the choroid, which are readily seen. The ophthalmologist can, by his eye examination recognize general diseases, such as exophthalmic goiter, syphilis, tuberculosis, kidney disease, brain disease, chronic spinal disease and others too numerous to mention, but the final treatment should be in the hands of the one best equipped for this purpose. The greatest advance in medicine in the last seventy-five years has been through this recognition of the value of divided labors.

CHONDROSARCOMA OF THE ORBIT REMOVED BY RESECTION OF THE OUTER ORBITAL WALL*

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N. S., aged fifty-two years, colored laborer, came to the Protestant Episcopal Hospital Dispensary on October 1, 1928, with the history of gradual and increasing proptosis of the right eye for the past two years, inflammation of the eye with great pain for the past two weeks and inability to sleep for the past week on account of ocular pain.

The appearance of the patient was very striking. The right eye was completely dislocated from its socket and only partly covered by the enormously stretched and hypertrophied upper and lower lids. Attempts at lifting the lid were resented by the patient, as this action was associated with much pain. The upper lid would readily slip behind the equator of the eyeball. The patient was quite adept in carefully replacing the eyelid so that it would cover a portion of the cornea. The movement of the lid was greatly restricted on account of the hypertrophy and pressure of the globe. The motion of the eye was limited to minute excursions. The cornea was very hazy, with an ulcerative process up and out from the corneal center. The pupil was contracted and no view of the fundus was obtainable. The vision was about 10/200, with normal vision in the fellow-eye. In the right orbit a large, firm mass with sharply defined outline could be readily felt, apparently filling the orbit and pressing the eyeball out of its socket.

Inasmuch as the man was badly nourished and weakened from the excessive pain, he was admitted to the hospital and strengthened for the removal of this growth. It was felt that the slowness of growth of the tumor presented a hope that it was not highly malignant. Upon admission to the hospital we learned the following:

The family history was negative. The present illness began about sixteen years ago, when the patient noticed a prominence of

* Read before the Ophthalmological Section of the College of Physicians, Philadelphia.

the right eye and was told by a physician that the right eye was larger than the left eye. This exophthalmos has gradually increased. For two years the lower lid has been everted. The vision of the right eye has been poor for fifteen years.

Operation, October 11, 1928, the outer orbital rim and the adjacent thin bone were removed with a powerful Ruskin rongeur forceps, so that the tumor could be easily felt and loosened from its connections. The encapsulated tumor was below the periosteum and was delivered through the orbital fissure by means of broad spatulæ. The eyeball was immediately drawn into the orbit. The deep temporal fascia was attached to the orbit periosteum by means of fine catgut sutures, a small rubber dam being used for drainage. The skin incision was sutured with silkworm gut and healed by first intention. There was no drainage for three days; then a small amount of pus exuded. This sinus healed in a few days. The corneal ulcer healed rapidly. The rest of the media also cleared so that a good view of the fundus was obtainable; the nerve was too gray, with a large area of choroiditis occupying the macular region and the region below the same. The ocular movements were restored completely in all directions and the lid was restored to normal function.

Pathological Report. Dr. C. Y. White reported the material to consist of a rounded tumor-like mass about the size of a walnut; consistency, firm; color, yellowish; surface, nodular. Sections show a fibrous-tissue capsule along one border. The mass of the tumor consists of sparsely-celled homogeneous tissue of cartilaginous structure. The cellular nuclei throughout this tissue are large, bladder-like and faintly staining; oval to oval; elongated in shape.

Throughout the section are numerous ducts lined with low, flat epithelium, generally in several layers; in many places the ducts are rounded to irregular in shape; in areas these ducts are dilated and cystic and contain a granular material staining slightly pink. Diagnosis: Chondrosarcoma.

In the classical Kroenlein operation we have a formidable procedure which requires much time in its performance. Many modifications have suggested. Dr. Harold Gifford, in the March, 1912, *Ophthalmic Record*, stated that he had removed the orbital bone, throwing away the fragments, thereby saving much time. He found no greater deformity than in the Kroenlein operation. Our case bears out his findings.

In brief, the operation consists of an incision, about 6 cm. long, beginning opposite the outer canthus, about 2 cm. away, and extending horizontally outward. After ligating the cut vessels and exposing the orbital ridge, a vertically placed curved incision is made along the orbital ridge through the periosteum. The periosteum is separated from both the orbit and temporal side with care for its preservation. With the orbital bone exposed, the thick horizontal ridge is first removed with a biting forceps, then as much of the thin bony wall is removed as may be necessary to give the needed outlet for the tumor. After the tumor is removed, the periosteal layer and the deep temporal fascia are united with catgut sutures, allowing a small opening for the small rubber-dam drain. The skin edges are then united. Care should be used to remove all bone fragments as made, in order to avoid a reaction due to the presence of a piece of bone acting as a foreign body. This operation can be readily done in thirty minutes and does not entail difficult situations.

The remarkable features of this case were the slow growth of an eventually large tumor in the orbit, apparently subperiosteal in its origin. The gradual extrusion of the eyeball was followed by a compensatory growth and stretching of the lids, which covered the eyeball until the latter was entirely out of the socket. The upper lid, then, no longer covered the cornea, and upon occasion would slip over the equator behind the eyeball.

The corneal ulcer healed rapidly, leaving a dense scar. The eye-ground showed a very large area of choroiditis in the macular region and below on the temporal side. Whether this was due to the stretching of the eyeball we cannot tell, as we have a history of poor vision for many years. The greatly extended lids gradually contracted in size and the ptosis has largely disappeared. The scar is not marked, but the outer canthus is drawn slightly to the temporal side in line of the excision.

THE DIAGNOSIS OF ACUTE MASTOIDITIS*

BY WILLIAM R. WATSON, M.D.
OTOLARYNGOLOGIST TO THE HOSPITAL

DESPITE all that has been published respecting acute mastoiditis, both in the text-books and in articles read by innumerable otolaryngologists, it still remains at times a most baffling pathological condition to diagnose with any degree of certainty. And this applies not only to the general practitioner but also to the specialist. Naturally, if either of these two groups of gentlemen are confronted with a frank "text-book" variety of mastoiditis, presenting all of the classical signs and symptoms, there is little excuse for either to fail in a correct opinion. I have reference more particularly to conditions wherein it requires almost a sixth sense to differentiate between an occult lesion simulating mastoiditis and mastoiditis itself. And, unfortunately, almost invariably, there is little time to spend over the diagnosis when the occasion for differentiation comes up, for the patient is usually in extreme and imminent peril. But someone is responsible for the determination of the diagnosis, and quickly, for in many cases any delay at all means the difference between life and death of the patient.

Although every physician is well acquainted with the signs and symptoms of simple acute mastoiditis, it might be well for purposes of further illustrating the text of my discourse to mention them briefly.

1. Pain; an almost constant symptom and of which the patient himself complains most: a dull, aching pain, frequently affecting the whole side of the head or located within the ear alone. Also, there is tenderness on pressure over the antrum or the tip of the mastoid and sometimes over the emissary vein.

* Read before a meeting of the Kensington Branch of the Philadelphia County Medical Society, November 6, 1925.

2. History of an aural discharge with sudden cessation, or the ear may be discharging profusely at the time of examination.

3. Temperature, typically around 100° to 101° F., unless there are complications, whereupon the fever becomes septic in character.

4. Redness and edema over the mastoid process and, in addition, in children especially, there may be fluctuation due to subperiosteal abscess, the cortex having become eroded and the pus breaking through.

5. Change of position of ear on affected side. It stands out further than the other ear.

6. On examination of the tympanic membrane it is found to be red, and, if not bulging, a perforation will be detected through which pus is coming from the tympanum.

7. There is a variable amount of deafness in the affected ear.

8. There is bulging of the posterosuperior wall of the external meatus. This sign, to me, is the most significant of all, not excepting the information that might be gleaned from a roentgen ray of the mastoid. This bulging occurs deep within the canal and very close to the tympanic membrane. It may be stated, however, that even this sign is at fault in the case of a mastoiditis of the tip.

9. There is a leukocytosis; in uncomplicated cases running from 10,000 to 15,000.

10. The roentgen-ray film shows a cloudiness over the affected mastoid, showing broken-down cell walls.

Now, it would appear to be perfectly simple to make a diagnosis if one were confronted by a patient presenting all of the symptoms I have just mentioned. But still, I have seen just such cases sent into the hospital with the diagnosis of acute mastoiditis which proved later to be no more than furunculosis of the external meatus.

For instance, in these cases of furunculosis to which I have just referred, there was pain; indeed, more subjective pain than in mastoiditis. There was swelling and edema back of the ear and tenderness on pressure, but with this difference: the tenderness over the mastoid in furunculosis is superficial, is exquisite on moving the ear up and down, and may be

elicited quite frequently by pressure in front of the ear over the tragus; whereas, the tenderness in mastoiditis is deep, and one may move the ear and press upon the tragus all he pleases without giving the patient pain. There was frequently a history of running ear, sometimes quite profuse, depending on the size of the furuncle that had burst. There was fever. There was at times fluctuation back of the ear, though I must confess that this is rare and is due to the furuncle breaking through the cartilaginous plate of the external meatus. I remember one case in the wards of the Episcopal Hospital of this type which I saw not so long ago. The woman had been sent into the hospital from the outside for a mastoidectomy, and after the abscess had been evacuated through a simple incision all her symptoms subsided. More often than not, the ear of the side affected with furunculosis stands out, sometimes further than in mastoiditis. Deafness in the affected ear is also common in furunculosis, as is also leukocytosis. Curious to relate, in my own experience at least, I have frequently had a roentgen-ray deceive me in furunculosis. Perhaps the patient might have had trouble in that ear years before, in which case the field over the mastoid would be hazy and show a paucity of intact cells over the affected side. Not until one comes to examine the tympanic membrane, in fact, does the examiner encounter his first and most enlightening point in the diagnosis. In the first place, if the furunculosis is in any way severe, the drum membrane will not be seen: the patient will flinch and complain bitterly at the entrance of the speculum. Moreover, the swelling is so great as to occlude the canal. In the second place, as the speculum is introduced, more often than not, the furuncle is seen discharging its pus, or, if it is not broken, the point is seen which, when touched with an applicator or the speculum, causes pain; whereupon, unless there is a coincidental mastoid involvement, the diagnosis is clear.

Another condition which simulates mastoiditis is a certain kind of mumps. The swelling in mumps, typically, is mostly in front and below the ear, and rarely behind. Notwithstanding this, last winter particularly, during the beginning of an epidemic of mumps, I was called into consultation several

times to determine the question as to whether certain cases were mastoiditis or not. There was swelling back of the ear, though not quite so far up as in mastoiditis or furunculosis. There was fever, of course, and tenderness on pressure, though not as acute as in mastoiditis and furunculosis. There was pain, especially on opening the jaw. With the exception of one of the patients, who had been suffering from an acute otitis, there was no discharge from the ear. The ear on the affected side did not stand out as in mastoiditis and furunculosis. There was no particular deafness. And, on examination of the eardrum it was normal in color and appearance, nor was there bulging either of the drum or posterosuperior wall of the canal. On these latter points I was able to rule out mastoiditis.

In the December number of *The Laryngoscope*, 1924, Lyman, of St. Louis, Mo., reports 4 cases operated upon for mastoiditis which were subsequently found to be suffering from sphenoiditis. All of these patients complained of pain in and around the affected ear, and 3 of them had recently been under treatment for purulent otitis media in the ear which had been operated upon. One of them had had 8 mastoidectomies without relief of pain, and all of them had been completely relieved by sphenoidectomies. I quote this article merely to indicate some of the obscure conditions which might simulate mastoiditis, but will not go into the differentiating points in the diagnosis, for the reason that these were regarded by the surgeons who operated as atypical cases. As you already know some of these so-called cases of atypical mastoiditis almost defy diagnosis. And, unfortunately, for this very reason, they are the most dangerous of all. I recall to mind a patient upon whom I operated in the Episcopal Hospital as the result of what might be called a "hunch;" certainly I had none of the classical signs and symptoms upon which to base a diagnosis. The patient was a child, aged three or four years, with little or no fever and no history of otitis media; the drum membrane appeared to be perfectly normal. There was no postauricular swelling or bulging in the posterosuperior wall of the canal; the roentgen-ray was by no means positive (and I pause here to remark that while in the majority of instances the roentgen-

ray is of inestimable benefit in the diagnosis of mastoiditis in adults, the same is not invariably the rule in infants, because the mastoid of infants is seldom developed beyond the antrum); the leukocytosis in this case was not particularly indicative of grave involvement; as I remember, the white count was in the neighborhood of nine or ten thousand; in fact, the only symptoms which induced me to consider operation were tenderness over the mastoid and the appearance of toxemia. At the operation, to show you how insidious these cases of atypical mastoiditis are, I had no sooner gouged through the cortex when a volume of pus gushed forth, apparently under pressure, and, on curetting the cavity, I found the lateral sinus exposed. Any delay at all in operating on this patient might have resulted in either a meningitis or brain abscess.

In lateral sinus thrombosis we have, besides all the signs and symptoms of acute mastoiditis, a series of additional symptoms as well as an intensification of those already existing, depending on the gravity and progress of the condition. Rigor, and an intense headache on the affected side I have always been inclined to look upon pessimistically. If, in addition, the temperature is fluctuating in character, and if there is an increased leukocytosis (especially in the polymorphonuclear count), and if there is marked tenderness below the tip of the mastoid extending down the neck, I should feel perfectly justified in immediately opening the lateral sinus. An early diagnosis and operation in these cases, even in the first premonitory signs, will do much toward preventing metastatic pneumonia, generalized septicemia or brain abscess.

These latter conditions are but later stages of lateral sinus thrombosis, and for the lack of time I will not dwell on them further, except to give you a brief picture of a case of general septicemia following mastoiditis treated in the Episcopal Hospital.

CASE REPORT. The patient, a boy, aged seventeen years, was sent in by an outside physician with the diagnosis of acute mastoiditis. He was gravely ill and so toxemic that he was scarcely conscious. There was no question as to the diagnosis and, on May 6, 1924, his mastoid was opened, revealing a most extensive necrosis of the bone in every direction. His lateral sinus was exposed during

the operation for about 2 cm., and since it appeared to be absolutely normal it was not opened. Indeed, it was considered that the toxemia originated from the necrosis. It was expected, therefore, that his symptoms would soon subside. Instead, however, he exhibited unmistakable symptoms of septicemia, and a few days later his blood culture was positive. Before waiting for the report a blood transfusion was given. At this time, some three or four days after his operation, he was practically moribund. The transfusion seemed to improve him considerably for a day, but he soon dropped back into his former state. Another transfusion was ordered and in the meantime, in consultation Dr. H. C. Deaver, suggested intravenous injections of mercurochrome, as a last resort; 30 cm. of a 1 per cent solution was administered and from that date he began his slow recovery; two or three more intravenous injections were given, which, while they caused a violent diarrhea, improved his condition in every case. It soon became evident, however, that he had but jumped from the frying pan into the fire. Metastatic abscesses began to appear over his whole body. No sooner was one evacuated when another would manifest itself. And now five and one-half years after his operation he lies in the ward, hopelessly crippled, both knees completely ankylosed, and still running pus from various places on his body.

The question naturally arises in your minds as to how this septicemia was acquired, since I have already mentioned the latter as being a later stage of lateral-sinus thrombosis. As a matter of fact, we finally concluded that the boy was suffering from septicemia when he was admitted, and, since the lateral sinus was found to be normal, the infection to the blood-stream must have traveled by some other route, perhaps by way of the lymphatics, or most likely by way of the emissary vein.

COMPLICATIONS OCCURRING IN MASTOIDITIS WITH A REPORT OF CASES

By CHARLES C. BIEDERT, M.D.
OTOLARYNGOLOGIST TO THE HOSPITAL

WHILE the diagnosis of acute mastoiditis is, as a rule, readily made, and the treatment usually simple and easy, the same is not true of those cases that are complicated, especially cases which have some intracranial complication which is often not recognized until after an operation has been done for the existing acute mastoiditis.

Not all the cases of suppurative middle-ear disease with symptoms of mastoid involvement should, in my opinion, come to operation; about 75 per cent of such cases, under proper conservative treatment, will clear up and get entirely well and will not suffer any recurrence. In some instances this occurs after an incision of the drum is made which gives better drainage, and in some cases it is due to sudden relief of blocking in the *aditus ad antrum*, coupled with discharge through the Eustachian tube. A case of this character was seen by me, a short time ago, in which a child of nine or ten years of age had, following a cold in the head, earache, rise of temperature to 102° or 103° F. with swelling and tenderness over the mastoid. This lasted for two or three days. There was redness of the drum membrane, but no discharge from the ear. An operation was urged, but the mother of the child begged for a twenty-four-hour delay until the father, who was a seafaring man, should return home. At the end of twenty-four hours the temperature returned to normal, the swelling and tenderness disappeared and the child got entirely well.

In acute mastoiditis it is often difficult to determine beforehand just how far the disease has gone, and we are often surprised on opening the mastoid cells to find that the necrosis

has extended further than we had expected from our study of the symptoms of the case.

It is, therefore, necessary to exercise rare diagnostic ability, coupled with good judgment, which comes only with long and large experience in these cases, to determine which should be immediately operated upon and which will recover without operation. In my mind there is just as much skill exercised, and just as much glory, attending the recovery of a patient without operation, where it is not needed, as there is attending the recovery of a patient after operation where it was necessary. It is the skilled surgeon who knows when to operate and when to withhold his hand.

When it is absolutely necessary to operate, do so without delay; but if there is a possible chance of the patient's recovering without operation, I think he should have that chance. This is the conservative view. I know there are others who take a more radical view of the case and advise operation in all cases where there are any mastoid symptoms. I am sure that, if this course is followed, many cases will be operated upon which would have recovered without operation had they been given the proper chance.

The complications most frequently met with in acute mastoiditis are brain abscess, lateral-sinus thrombosis, meningitis and paralysis of the facial nerve.

As before stated, the symptoms of intracranial complications are often not prominent and are frequently not recognized until after an operation has been done for mastoiditis. Whenever the temperature remains high after a mastoid operation, or if we have a sudden rise after an interval of normal temperature, we usually suspect some intracranial complication.

In brain abscess the classical text-book description of sub-normal temperature, slow pulse, constipation and dilated pupils is seldom met with. The temperature is just as likely to be elevated (around 102° or 103° F.) with irregularity of the pupils and in some cases choked disk. The most frequent sites of abscess are the temporosphenoidal lobe and the cerebellum. It is rarely that we have any localizing symptoms unless the abscess should occur over the motor area. In most

cases it is like hunting for a needle in a haystack to locate the pus.

The following case is an example of brain abscess:

CASE I.—A child, aged thirteen months, was admitted to the Episcopal Hospital with the history that two weeks before admission there had been pain in the left ear, following a cold in the head. The ear then began to discharge and this was followed in two days by swelling over the mastoid.

On admission the child appeared quite sick, temperature, 102° F., and there was a fluctuating swelling over the left mastoid. An operation was done at once, a large amount of pus evacuated and the mastoid cells broken down. The cells were all curetted and through-and-through drainage established from the middle ear. After the operation the child did not do well; the temperature remained high, pulse and respirations were rapid, and bronchopneumonia was suspected. The child remained in pretty much the same condition for four weeks. The mastoid wound healed nicely. It was then suggested that the anterior fontanelle be tapped to obtain spinal fluid for examination. This was done and pure pus was drawn into the syringe. The child was then again operated upon, the skull trephined over the temporosphenoidal area and exploration of the brain made in all directions, but no pus was found. The child was in greatly weakened condition from its long illness and the operation had to be abandoned, the child dying shortly afterward. No autopsy was allowed.

This case exhibits some of the difficulties encountered in the diagnosis and location of the abscess.

In lateral sinus thrombosis the symptoms are usually more pronounced and definite. In these cases we have the typical septic temperature curve; the steeped chart, as it has been designated. Usually, following a chill, the temperature will rise to 103° or 104° F., and then rapidly falls to subnormal, sometimes accompanied by a sweat. This may be repeated every day or two, according to the severity of the infection.

The following case is an example of this complication:

CASE II.—An Italian, aged nineteen years, was admitted to the medical ward of the hospital with lobar pneumonia, during the course of which he complained of earache. The drum was found

bulging and was promptly incised. The ear condition apparently cleared up and he recovered from his pneumonia.

Two weeks after the first attack of earache, he again complained of pain in the ear, and the temperature rose and after a day or two assumed the typical septic type. Examination of the ear showed redness of the drum, no bulging and no discharge. A diagnosis of possible brain abscess was made and operation was recommended.

The mastoid antrum was opened, but very little pus found. The temporosphenoidal lobe of the brain was exposed through the opening in the mastoid antrum, but no pus was found.

The lateral sinus was then exposed and found to be thrombosed. The sinus was opened and broken down, clot and pus removed and the sinus curetted in both directions until free bleeding occurred. The sinus was then packed. The patient made an uneventful recovery and was able to leave the hospital in three weeks.

In meningitis complicating middle-ear disease we usually have severe headache, high fever with rigidity of the muscles of the neck, Kernig's sign and Babinski's reflex. In one case which came under our notice, upon making a spinal puncture, pure pus was obtained, which showed a pure culture of pneumococcus. The patient was given two doses of antipneumococcic serum into the spinal canal, the pus was drawn off every day and the patient finally made a good recovery. I have always thought the pneumococcus in this case must have been of a nonvirulent type.

Paralysis of the facial nerve occurring as a complication of mastoiditis may be due to destruction of the nerve as it passes through the aqueductus Fallopii, or the paralysis may be due to pressure on the nerve as it leaves the stylomastoid foramen, occurring in cases of mastoiditis with perforation of the tip of the mastoid process, the so-called Bezold's abscess.

The following is an example of this:

CASE III.—An adult male was admitted to the hospital with the history that, three weeks before, he had severe earache following a cold in the head. He was treated by his family physician and, one week before admission, swelling and pain over the mastoid developed.

On admission, he was quite tender over the tip of the mastoid

and there was swelling and tenderness in the muscles of the neck. There was a complete Bell's palsy on the affected side.

At the operation, the mastoid antrum contained pus. There was a large cell at the tip of the mastoid, with a perforation, and pus invading the tissues of the neck. Drainage was provided and he made an uneventful recovery in about five weeks' time, the facial palsy having cleared up as the swelling in the neck disappeared.

If the paralysis is due to involvement of the nerve as it passes through the aqueductus Fallopii, it is usually permanent, because the injury to the nerve in this position is due to the extension of the necrotic process and the nerve is destroyed along with it. It is here that the nerve is most likely to be injured in the operation for mastoiditis.

APHONIA:

REPORT OF THREE CASES*

BY WILLIAM R. WATSON, M.D.
OTOLARYNGOLOGIST TO THE HOSPITAL

APHONIA is defined by Webster as being a "loss of voice or vocal utterance, due to disorder of the vocal cords." Moreover, to enlarge on this simple definition, vocal utterance is defined as an "element of speech consisting of pure vocal tone." Thus, notwithstanding an individual by whispering may express himself in words and make himself understood from a variable distance away, he has nevertheless lost his voice and is suffering from what is termed aphonia. There are certain aphasias, however, whereof it is said the patient "is unable to speak." But no one ever thinks of calling this aphonia; nor does vocal utterance in these particular cases depend on the cords.

Aphonia is a condition that so commonly confronts the modern laryngologist that apparently it is scarcely deemed worth while reporting. This subject was more popular in the past, however, for the older literature is full of it. But, curiously enough, I found that rarely indeed was this affection attributed to physical causes. Almost invariably, the case reports were of patients suffering from "functional aphonia."

ETIOLOGY. The study of the particular etiology in certain cases that I have attended has been well worth the time I have spent on them, and I have chosen a group of three out of a number that have appeared in the clinics of the Episcopal Hospital as offering sufficient interest to report, especially as one of them offers a rebuttal, at least tentatively, to the

* Read before the Otological Section of the College of Physicians, Philadelphia, April 21, 1926. Reprinted from the Archives of Otolaryngology, 1926, vi, 46.

claim of certain laryngologists that primary tuberculosis of the larynx cannot exist.

There are several exhaustive classifications giving the causes of aphonia. I will not repeat them, but will mention a few of the more common causes, more or less in the order of their frequency so far as my experience has taught me.

1. Tuberculosis is the commonest cause; that is, of the comparatively permanent aphonias. As a matter of fact, acute catarrhal laryngitis is the most frequent cause of all; but as this condition is limited to only several days' duration, it does not come within the purview of this paper. Tuberculosis may act locally on the cord or ventricular band to cause aphonia, or, by distortion of the arytenoids by means of edema or tumorous tubercles, may prevent the cord from approximating.

2. Syphilis is a common cause, according to my experience. It may act locally or indirectly; indirectly by pressure of a syphilitic tumor on the cortical centers, or on the trunk of the tenth cranial nerve, or on the recurrent laryngeal, or even on the terminal endings of the latter in the larynx, though this is rare. Unusual conditions resulting from syphilis are the aphonias noted in locomotor ataxia and paresis of the cortical centers from a broken-down or softening gumma.

3. Benign or malignant growths around or on the vocal cords are a fairly frequent cause, though in the majority of these cases the patients do not wholly lose their voices. They usually speak in a husky or whistling tone, which is so characteristic that the condition is often diagnosed without examination. The ordinary papilloma is the most frequent offender.

4. Paralysis of the intrinsic muscles of the larynx through neuritis of the controlling nerves caused by a focal infection; by pressure on the trunk of the pneumogastric or recurrent laryngeal nerves by pericardial or pleural effusions and mediastinal growths, or by aneurysm of the arch of the aorta, or even by a hypertrophied thyroid gland, as well as by enlarged glands in the neck or tumors of the esophagus.

5. Paralysis of the cords or intrinsic muscles in certain forms of infectious diseases, such as typhoid fever and diphtheria, which, while not permanent, are at times of several

weeks' duration. The aphonias found in the exanthematous diseases are as evanescent as those in acute catarrhal laryngitis.

6. A rare cause of aphonia would be embolism, causing an infarct in the cortex of the brain or apoplexy in the same area, or a tumor (mild or malignant) pressing on the cortical centers governing the muscles of the larynx.

7. Shock, from concussion of the brain, as from a blow on the head, providing it causes incoördination of the centers in the cortex; or from a fractured skull lacerating the trunk of the tenth nerve; or from a lacerated wound in the neck, or an incised wound, such as would be made in a thyroidectomy, causing trauma of the motor nerves of the larynx.

Besides the organic causes for aphonia, the latter condition may be found in almost any neurosis.

The 3 cases which I desire to report are as follows:

REPORT OF CASES. CASE I.—E. D., a bartender, aged sixty-two years, was admitted to the wards of the Episcopal Hospital on May 13, 1925. The family history was negative.

Several months before coming to the clinic he was becoming hoarse for no apparent reason, and a short time later lost his voice entirely. There was no pain in the throat either on swallowing or otherwise. He coughed seldom and not much at any time. He had lost little weight. No fever or nightsweats were present at any time. In other words, the only symptom from which he suffered was the loss of voice.

The patient appeared in the dispensary, giving the history as stated in the foregoing. Indirect laryngoscopy revealed a swelling in the posterior commissure extending up between the arytenoids, which held the cords apart and so fixed as to prevent them from approximating, although there was distinct though somewhat limited abduction on inspiration. The tumor was small, flat and rough-looking; that is to say it had not the smooth and velvety appearance of mucous membrane, and no ulceration could be detected. The epiglottis was free from any defect and the arytenoids were of a normal color and not enlarged. No glandular enlargements could be found in the neck or elsewhere. The vocal cords were of a normal color, except perhaps that they were slightly pink in the part contiguous to the tumor.

A Wassermann test was made, which proved negative. Although the condition had already been tentatively diagnosed as malignant,

the patient was sent to the chest clinic for a careful study of his lungs and sputum. Several exhaustive examinations were made, including the usual frequent roentgen-ray examinations of his chest, but no evidence of pulmonary tuberculosis was found.

Therefore, since no subjective symptoms of tuberculosis could be found, and especially after the assurance by the chest clinic, and also on account of a vigorous denial of venereal history as well as a negative Wassermann reaction, the laryngologic section of the hospital felt fairly well justified in making their first diagnosis of malignancy definite. His age, as well as the appearance of the tumor, were factors in this conclusion. He was sent to the hospital merely to substantiate this diagnosis by biopsy.

A specimen was collected by direct laryngoscopy, and sent to the laboratory with the expectation of a diagnosis of carcinoma, but no cells simulating either benign or malignant tumor were discovered. Typical tubercles were found, however, though a search after staining the sections for acid-fast bacilli was ineffectual.

This would almost appear, therefore, to be primary tuberculosis of the larynx. I belong to the group of laryngologists who believe that there is no such thing as primary tuberculosis of the larynx. At any rate, the condition of this patient comes closer to this diagnosis than any other I have ever seen. I saw this patient frequently thereafter, the last time on September 22, and his symptoms had not changed. He felt well and had lost a little weight during the summer, but had regained nearly all of it since. He coughed no more than he did before. There had been no fever or nightsweats. He had had no pain on eating and no discomfort elsewhere except aphonia. On examination of his larynx from time to time, it was observed that the lesion in his posterior commissure had extended to the ventricular band on the right side. The latter was so swollen that the cord on that side could not be seen.

CASE II.—R. L., a negro, aged twenty years, a laborer, had practically the same lack of symptoms as in Case I, that is, there was no discomfort other than loss of voice. Moreover, there was no loss in weight, no elevation of temperature when he appeared in the dispensary, no nightsweats, and no cough to speak of. The family history was negative.

This patient appeared in the clinic of the Episcopal Hospital, May 27, 1925, with the statement that he had begun to get hoarse two years before when a fishbone stuck in his throat. He had succeeded in coughing up the fishbone, but the hoarseness persisted. For several months he has been speaking only in a whisper.

Examination disclosed enormous tonsils and indirect laryngoscopy a practically normal larynx, with the exception of a right ventricular band so swollen as to eclipse totally the vocal cord on that side; moreover, this band apparently was bound down to the vocal cord. There was normal abduction of the left cord on inspiration, but the right cord could not be seen. There was no edematous swelling of the epiglottis or the arytenoids, nor could the color of the mucous membrane of the larynx be called abnormal. A gland or two of the anterior chain in his neck could be felt, which, however, was attributed to the infected tonsils.

Two Wassermann tests, and even an additional provocative Wassermann test, were negative. In the meantime, in the forlorn hope that they might have some inflammatory effect on the larynx, the tonsils were removed. This had no effect on either the phonation or the swelling of the ventricular band.

This patient was also sent to the chest clinic, and while no microscopic evidence was found in the several specimens of sputum, nor were any definite signs found by physical examination, it was discovered that he invariably ran a daily temperature of 99.3 and 99.4° F., which seemed to be sufficient to justify a diagnosis of incipient pulmonary tuberculosis.

During the last days of September, 1925, we were able to locate this patient as a laborer on the subway, but could not get him to come to the clinic. He sent back word that he was feeling fine, but he was still speaking in a whisper.

CASE III.—A. N., a white woman, aged sixty years, was admitted to the medical wards of the Episcopal Hospital on May 17, 1925, for study. Her only symptoms were aphonia and cough, and her family history was negative. Her health had been good except for cough and colds. Twenty years before presentation she had suffered from abscesses in both legs. She became hoarse every time she acquired a cold, which would persist for weeks. She had never expectorated blood. She perspired a good deal at night; there were no dyspnea or headaches, but she had vertigo at times. There was no pain on swallowing. She was married when she was forty years of age, and was never pregnant.

Her present illness started with a bad cold in the chest about ten weeks prior to examination, with productive coughing. The sputum was yellowish, thick and tenacious, but there was no blood. There was no pain in the chest, no chills or fever.

Physical examination on admittance showed nothing of importance.

The patient was referred to the laryngologic service of the hospital, and, on examination by mirror, a paralysis of the right vocal cord was revealed. A Wassermann test and a roentgen-ray study of her chest in the region of the right recurrent laryngeal nerve were suggested. The Wassermann reaction was 4+. The roentgen-ray report by Dr. Ralph Bromer was as follows: "Extreme thickening of the hilum in both lungs, extending into the apexes, is present. Nothing is found with reference to aneurysm of the aorta or mediastinal effusions or tumors. It is possible that the appearance of the lungs may be due either to hilum tuberculosis or to syphilis."

Examination of the blood and urine was uninteresting. The sputum was normal.

Neurologic study by Dr. George Wilson disclosed the following: "Bilateral involvement of the eleventh and twelfth, and unilateral involvement of the tenth cranial nerves, central in origin."

The patient was given several intravenous doses of arsphenamine, with apparently no result so far as her phonation was concerned. This patient was seen again on October 29, and it was noted that the right cord had slightly increased in function, but was still badly paralyzed. The voice was better. The whisper had become a voice, hoarse and croaking as it was. At that time the patient had received fifteen intravenous applications of arsphenamine in all.

COMMENT. As I have mentioned, this patient had been suffering from aphonia for ten weeks, and, if we except the cough, from nothing else. The appearance of the larynx was perfectly normal; there was not the slightest sign of either acute or chronic inflammation or edema, despite her cough. The only abnormality, in fact, was the paralyzed cord, which, while not exactly in the so-called cadaveric position, stood far enough apart from the other cord so that attempted phonation was ineffectual. The question is, therefore, was she suffering from a functional aphonia at this time, or did she actually have paralysis of the cord from a central syphilitic lesion? It would almost seem that if one could forget the 4+ Wassermann reaction and the neurologic report, as well as the patient's apparent recovery by intravenous medication, we might think of it as being functional in character.

In this respect it might be interesting to repeat the opinions of certain members of the Royal Society of Medicine, London, with reference to unilateral paralysis of the cord, given in a

recent abstract of the proceedings of the latter society. The case which caused the discussion was that of a man, aged fifty-six years, with immobility of the cord for eighteen months, which was reported by M. Vlasto, F.R.C.S. The history was as follows:

The subject was a painter, who had been hoarse for eighteen months. The laryngeal parts were congested. The left false cord was so swollen that the true cord could not be seen. The Wassermann reaction was negative. The chest was normal, and no dysphagia was present. Shortly after the first examination some of the laryngeal swelling subsided, and then it became apparent that the left cord was fixed. The condition had not changed in six months. Opinion was invited.

In the discussion, Mr. Herbert Tilley referred to a paper he had published in 1918 in which he had followed up the after-histories of 23 cases of paralysis of the cords in private practice during twenty years. No reply was received from 10 patients; 7 had completely regained the function of the cords; 3 had died, and 3 had continued in the same condition as when first seen. The fatal cases were due to aneurysm of the aorta and to malignant disease of the mediastinum and gullet. The 7 recoveries were probably due to ankylosis of the crico-artenoid joint.

Sir William Milligan thought that many of these cases were functional, though this went unrecognized when only one cord was affected. He had seen several cases thought to be due to organic disease, and when they were presented several years later he found that they had regained their function without treatment, which suggested that they were almost certainly functional. The possibility of a unilaterally paralyzed cord being of functional origin should not be overlooked.

Sir St. Clair Thomson said that possibly there was functional paresis of one cord. He had watched cases, and sometimes could not make up his mind whether there was complete mobility of a cord or not. He had had 11 cases of paralysis of the cord in tuberculous patients with nothing wrong in their larynx. In 5 of the 11 there was recovery of movement. Of the remainder, 1 or 2 disappeared from observation, and in 2 or 3 the condition had become permanent after several years.

CUTANEOUS NEUROMA*

BY JOHN B. LUDY, M.D.
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THE purpose of this paper is to submit a case presenting all the features essential to the diagnosis of cutaneous neuroma occurring in a patient having tuberculosis.

By virtue of the rarity of cases observed, neuroma cutis has meant many things to many persons. Wood's¹ "subcutaneous painful tubercles," Rump's² fibroid tumors of subcutaneous nerves, the so-called false neuromas of Virchow³ and Darnell's⁴ plexiform neuromas have all been reported as cases of neuroma cutis. The term itself, however, limits us to those affections which deal with growths containing abnormal nerve elements and having their location and development in the true skin. The subcutaneous nodules mentioned, together with the "neurofibromas" of Recklinghausen and the nodules of nerve tissue springing from amputated nerves will, therefore, not be included in this title.

Duhring,⁵ Kosinski,⁶ Heidingsfeld,⁷ Little⁸ and Duemling⁹ have each reported a separate case which has been accepted as a true case of neuroma cutis.

In addition, Verneuil¹⁰ reported a dissecting-room case of myoma cutis in which nerve fibers were present in such abundance that the diagnosis of neuroma could be entertained almost as well as that of myoma.

In 1893, Cavafy reported to the Dermatological Society of London a case of extremely tender multiple tumors on the back of a woman. This, in Hartzell's opinion, was neuroma cutis.

The authors of the *American Text-book of Pathology*¹¹ believe that "the multiple dermal neuromas found by Knauss¹² in children can be included in the list of neuroma cutis."

* Read before the dermatological section of the American Medical Association, July 12, 1929.

Heidingsfeld notes that "of the 14 cases of well-defined myoma cutis which have been reported, 9 were painful, and in some of the cases nerve abnormalities were noted."

Conceding these possibly dozen additions to the 5 cases more accurately reported, the affection may still be one of the rarest known to dermatology.

Duhring's report and description were published in 1873. His clinical diagnosis was Tuberculide. The lesions were discretely and irregular disseminated over the left arm and shoulder of a man aged seventy years, and consisted of split-pea sized, firm, flattened tubercles, purplish in color and firmly incorporated in the skin. Paroxysmal pain constituted the distressing feature. This, however, did not manifest itself until three years after the tubercles had begun to form. Microscopic examination¹³ revealed the growth to be made up of a connective-tissue stroma interwoven with numerous nonmedullated nerve fibers.

Kosinski's case was very similar to Duhring's, with the lesions limited to one leg and buttock and later painful. His clinical diagnosis was also tuberculide and the diagnosis of neuroma was made by the microscope.

Little⁸ does not record a clinical diagnosis, but the description of the lesions would suggest tuberculide: "hundreds of small, bluish-red, hard nodules, split-pea sized, in the substance of the skin." Section showed the bulk of the tumors to be made up of nerves.

Heidingsfeld's⁷ case presented a solitary encapsulated nodule, developing subsequent to an injury and painless for twenty years. His clinical diagnosis was myoma and the microscopic diagnosis, neuroendothelioma.

Duemling's⁹ case was a single palm-sized lesion of the breast, entirely painless for the three months of its duration. The clinical diagnosis was spontaneous keloid, or "cancer en cuirasse." Microscopic diagnosis, neuroma.

CASE REPORT. M. J., a colored female, aged thirty-seven years, who stated that as a young girl she was greatly troubled with distressing and prolonged "colds," which were intermittently present between the ages of eighteen and twenty-five years; that

thereafter she gained in weight and considered herself well until four years ago when the lesions responsible for her later admission to the Episcopal Hospital made their appearance.

Physical Examination. X-ray of the chest shows cottony infiltration of the left apex, suggestive of an old inflammatory condition, probably tuberculous in origin. Dense, opaque shadows in the lower portion of the right thorax, with costophrenic angle obscured, suggest a thickened pleura with fluid.

The skin manifestations (Figs. 1 and 2) were limited to the extremities. The face, trunk and thighs are entirely free. On each extremity from fifteen to twenty deeply pigmented, discrete, split-pea to hazlenut sized, firm, flattened tubercles are noted. Several show umbilication. They are firmly incorporated in the skin and are diffusely distributed, not following any nerve trunk.

They began to appear four years ago, new lesions appearing gradually. Except for occasional periods of mild itching, they were symptomless until one year ago, when she came under observation. At that time she referred to them as having "spells of severe itching," and producing knifelike pain and being painful and tender on handling, and again, at other times, they were neither tender nor painful.

The von Pirquet test is positive; the blood and spinal Wassermann are negative, complete blood-count shows 4,320,000 erythrocytes, 6200 leukocytes, 80 per cent hemoglobin, 57 per cent neutrophils, 35 per cent lymphocytes, 5 per cent large mononuclears, 1 per cent transitional and 2 per cent eosinophiles per hundred.

The patient has been given old tuberculin at irregular intervals. recent x-ray studies show improvement of her chest; no new lesions have appeared during the last two months and she believes her attacks of paroxysmal pain to be less severe and more infrequent.

Microscopic Report. The stratum corneum, the stratum granulosum and the prickle-cell layer are markedly hyperplastic (Fig. 3).

The compelling feature of the corium is the presence of numerous ill-defined strands of medullated nerve trunks which extend in all directions through the subpapillary parts—some parallel to the surface, others appearing in transverse section. Particularly striking are those strands which extend in a perpendicular direction as far as the tips of the papillæ. There are no inflammatory infiltrates actually within the nerve trunks, but there are comparatively dense accumulations of lymphocytes around them (Fig. 4). The nerve fibers are traceable into the deeper portions of the corium, but there is much less here than in the subpapillary parts. The large round-cell infiltrate is also to be found around the blood-



FIG. 1



FIG. 2

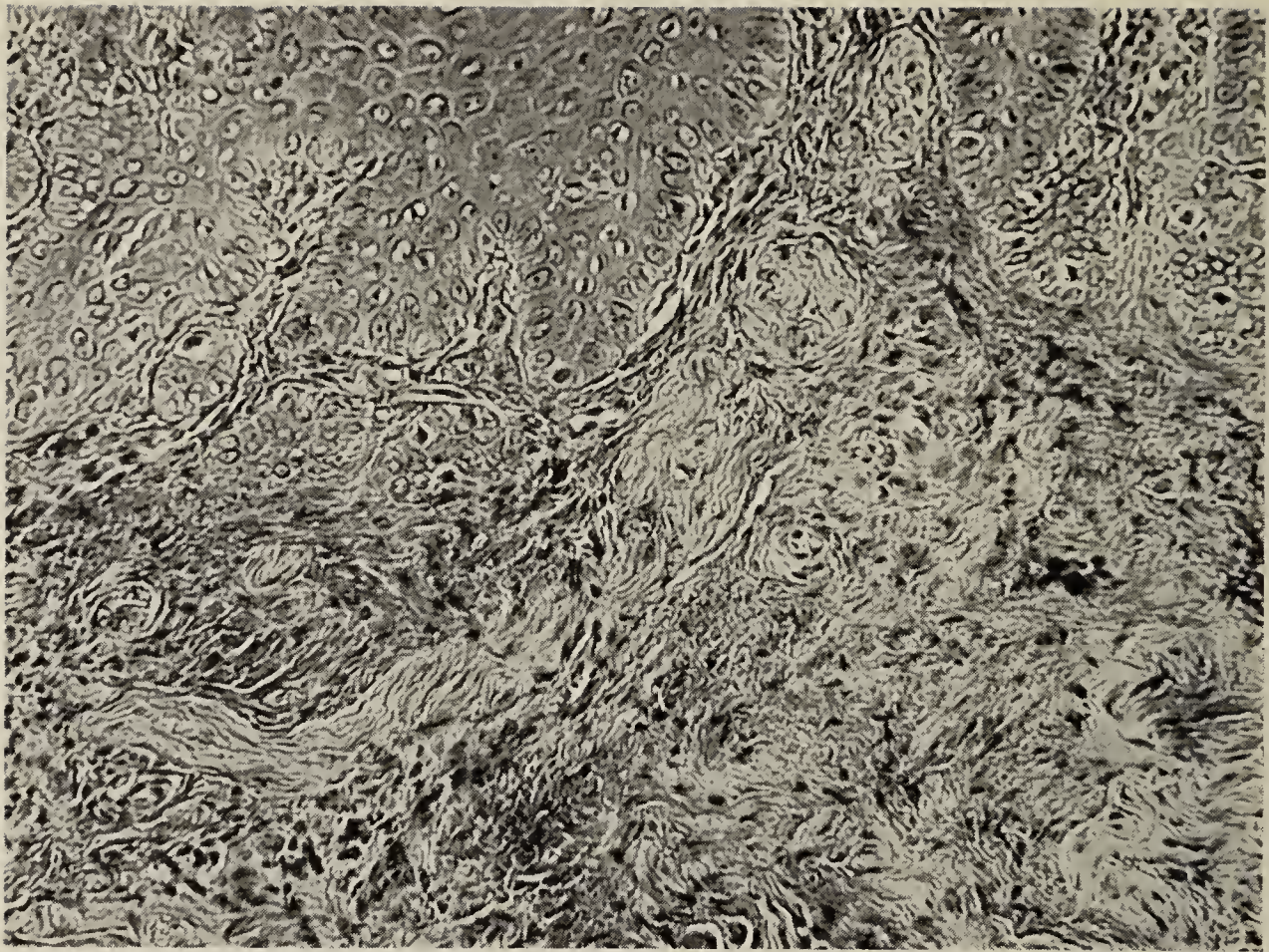


FIG. 3



FIG. 4

vessels. It is significant, however, that the densest accumulations occur around the nerve tissue, and that the nerve elements occur precisely under those parts of the epiderm which are the most markedly hyperplastic. The nerve distribution is not a peculiarity of the anatomy of the entire skin of the patient. Large numbers of fibroblasts, congregated in the perivascular lymph spaces, may well be expressions of a diffuse granulomatous infiltration.

Van Gieson's stain distinguished the yellowish-brown involuntary muscle bundles deeper down from the strongly red nerve trunks. The fibrillar portion of the nerve trunk stained pink; the medullary sheath stained yellow. Ziehl-Neelsen method demonstrated no acid-fast organisms.

Neuroma cutis, therefore, would imply a new growth located intimately within the skin, most frequently assuming the character and distribution of tuberculides but occasionally solitary and variously sized, commonly tender and painful and containing abnormal nerve elements.

Thus far the diagnosis has been made only by the microscope. Heidingsfeld comments that "all excessively and persistently painful lesions of the skin, in which the pain is of a spontaneously paroxysmal or easily induced character, warrant careful technical examinations for nerve fibers." The essential pathological lesion is located in the corium and consists of a meshwork of nerve fibers, medullated or non-medullated, supported by a connective-tissue stroma. There may or may not be present other elements, such as blood-vessels, lymphatics, elastic tissue, unstriated muscle and various cells indicative of a granulomatous stimulus.

The search for etiological sequence is concentrated on the abnormal nervous tissue. Are the nerve elements found in these lesions dislocations of normal nerve trunks? *The American Text-book of Pathology*¹¹ points out that certain tumors "may take their origin from the endoneurium, pushing aside the nerve fibers until they become widely separated," and that "it should be clear, therefore, why a histological study of the neurofibromas as found in the skin and along the course of peripheral nerves should show gradations relative to the nerve fibers, which are sometimes abundant, again

isolated or even absent, and why the fibers vary from apparently normal medullated fibers to naked axis cylinders or axis cylinders variously formed."

Louis Heitzmann¹⁴ also questions "whether the nerve fibers in neuromata cutis are newly formed. If the internal nerve sheath be increased, the innumerable nerve fibers are pushed apart and thus may give the impression of constituting a true neuroma."

In the present case, moreover, it appears as though there may have been a larger nerve trunk, but that it has become split up into separate subfasciculi, a dendritic separation and distribution of one of the nerve filaments in the skin. Sufficient evidence, therefore, seems to be at hand to warrant the conclusion that the nerve elements may represent the disintegration of some parent nerve trunk.

On the other hand, a distinction should be made between tumors taking their origin from the endoneurium and nodules such as tuberculides, which depend upon a toxic stimulus producing focal granulomatous reactions. The fibroid and plexiform tumor, the neurofibromas of Recklinghausen, painless embryonic connective-tissue new growths, are accredited with hereditary characteristics and with arising from embryonic rests. They are tumors in the exact meaning of the word. The nodules under discussion are the result of acquired irritants, irritants which produce degenerative and proliferative reactions, and pain when contact is made with sensory nerves. Heidingsfeld very pertinently remarks that "the explanation that the pain in myoma cutis is due to mechanical pressure scarcely suffices. Otherwise all growths in the cutis would give rise to similar symptoms."

"As is well known, the nerves are nothing more than axis cylinders, that is, the expansile portion of the nerve cell or soma. In the presence of toxins, poisonous and traumatic actions, the nerve is much more active than the central cell. Thus, while the soma and dendrite possess only slight powers of regeneration, reacting rather by internal or structural metamorphosis, the axis cylinders respond to stimuli not only by structural or intraprotoplasmic metamorphoses but also by external transformations.¹⁵

That proliferation of nerve tissue as a result of irritant actions may be of wider scope than is generally recognized, is suggested again by Heidingsfeld, when he notes the "marked proliferation of erector pilorum in some of the chronic dermatoses, such as pityriasis rubra pilaris, which are attended with persistently creepy and chilly sensations." He comments that such hypertrophies "can readily be considered an effect rather than a cause, and, if this be true, no cutaneous abnormality would be more conducive to involuntary muscle proliferation than a neuroma."

It should be proper, therefore, to question whether the meshworks of nerve fibers found in neuroma cutis do not represent proliferations of nerve tissue. Our impression is that these nerve elements may be found to represent a true proliferation induced by some toxic or traumatic action.

SUMMARY. 1. A case is submitted of painful lesions, diagnosed clinically as tuberculide, and demonstrated by microscope to be neuroma cutis.

2. Painful lesions in which the pain is of paroxysmal type warrant careful technical search for nerve fibers.

3. A distinction is to be made between tumors in the strict sense of the word and nodules belonging to the category of granuloma.

4. It is our belief that the nerve elements found in neuroma cutis represent proliferation of nerve elements.

BIBLIOGRAPHY

1. Wood, W.: Trans. Med. Chir. Soc., Edinburgh, 1829, iii, 29.
2. Rump: Arch. f. path. Anat. u. Phys., 1880, lxxx, 177.
3. Virchow: Die Kraakhaften Geschwülste, 1864, ii, 150.
4. Darnell, W. E.: Multiple Neuromata of the Skin, Jour. Am. Med. Assoc., 1906, xxxvii, 1810.
5. Duhring, L. A.: Painful Neuroma of the Skin, Am. Jour. Med. Sci., 1873, lxvi, 413.
6. Kosinski: Centralbl. f. Chir., 1874.
7. Heidingsfeld, M. L.: Jour. Am. Med. Assoc., 1913, lxi, 435.
8. Little, E. G.: British Jour. Dermatology, 1919, xxxi, 102. Also Proceedings Royal Society Med., Section Derm., 1919, p. 35.
9. Duemling, W. W.: Arch. Derm. and Syph., 1929, xix, 226.
10. Verneuil Am. Jour. Med. Sci., July, 1874.
11. American Text Book of Pathology, pages 175 and 194.
12. Knauss: Virchow's Archives, 1898, cliii, 29.
13. deSchweinitz, G. E.: Am. Jour. Med. Sci., 1881, lxxxii, 435.
14. Heitzmann, Louis: In P. A. Morrow's System of Urology, Syph. and Derm., Appleton & Co., N. Y., 1894.
15. Cajal: Regeneration and Degeneration of the Nervous System.

GENERALIZED ALOPECIA

REPORT OF THREE CASES, WITH ONE NECROPSY*

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AND

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THE universal loss of hair from the body has occasionally been described in medical literature and, although no constant etiologic factor is present in such a condition, various causes have been ascribed, most of them on theoretical grounds. Alopecia areata infrequently produces a universal falling out of the hair. In syphilis loss of hair occurs, but it is seldom complete; Chambers,¹ however, in 1901 reported the case of a woman, previously treated for interstitial keratitis, whose hair fell out in patches when she was five years of age, but it returned in a short time. At the age of twelve, her hair began to fall out again, and with the exception of two small, fine ones on the anterior portion of the scalp, she had no hair on the entire body at the age of twenty. Arnett² cited a case which he thought was due to syphilis; the patient was twenty-six years of age, and had lost his hair eight years previously; the Wassermann reaction was strongly positive, and at the end of seventeen months of antisyphilitic treatment the hair was entirely restored. This patient had had two previous attacks one at the age of six and the second at the age of twelve years, and in both the hair returned with the employment of only local measures. Thompson's³ case occurred in a man, aged twenty-three years, who, two months after the appearance of a chancre, lost all his hair and four

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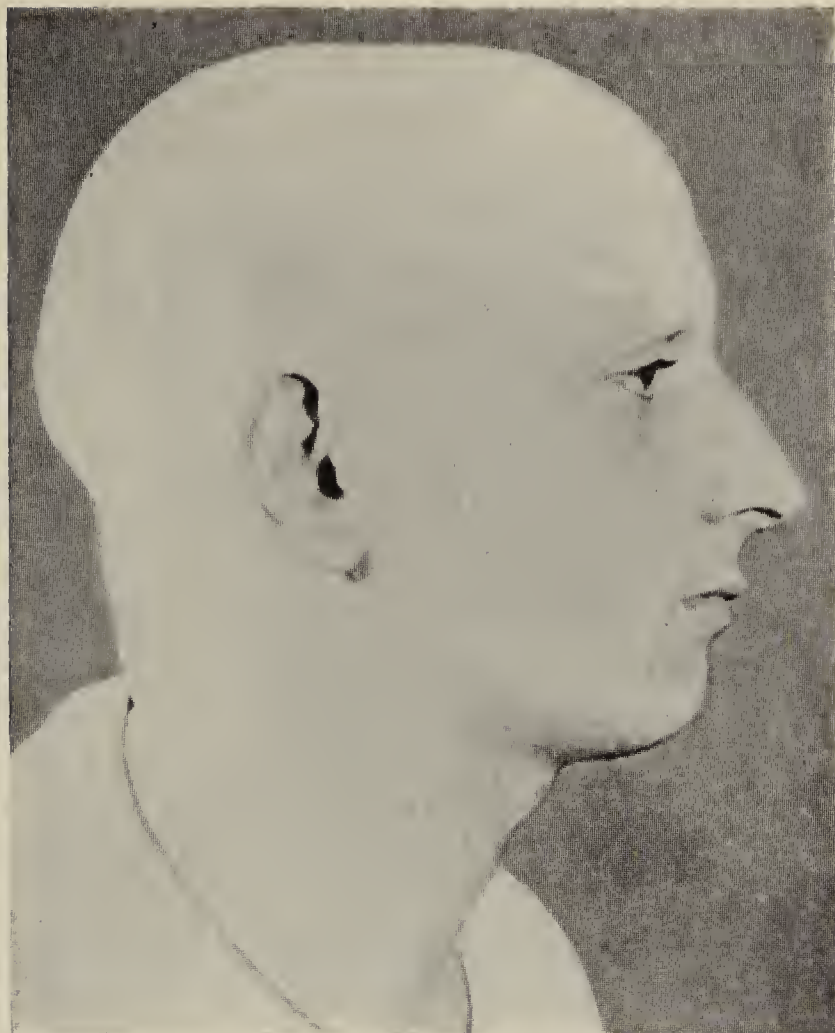


FIG. 1.—Case II. Loss of hair from scalp and eyebrows as result of alopecia.

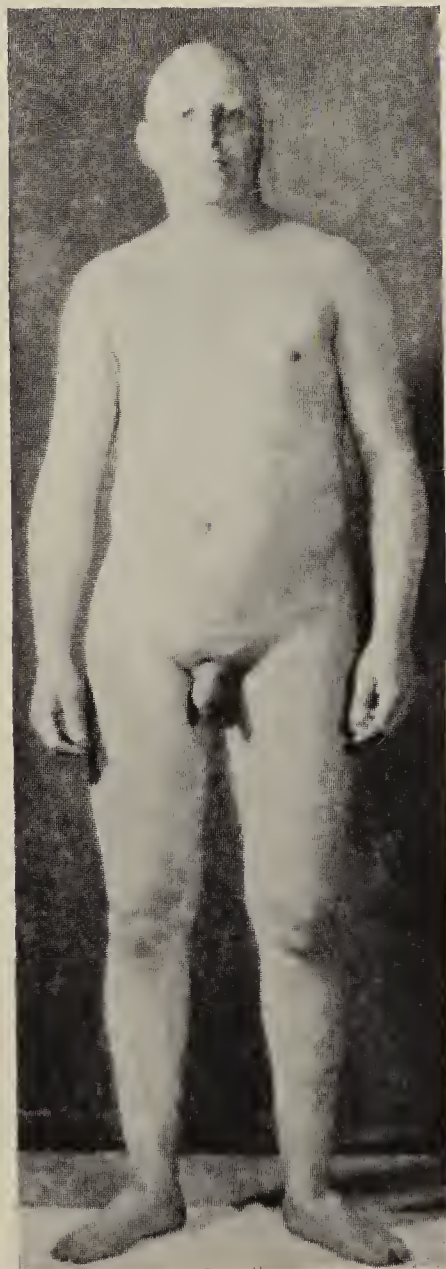


FIG. 2.—Case II. Complete loss of body hair.



FIG. 3.—Case III. Loss of hair on head and in axilla.



FIG. 4.—Case III. Loss of body hair.

years later there was no return. Alfred⁴ saw a case of generalized alopecia, which apparently had resulted from scarlet fever in early childhood. Sajous⁵ reported the case of a woman, aged thirty years, otherwise in good health, who in less than a week lost the hair of the entire body, not even an eyelash remaining. Different lines of treatment were used over a period of two years, and the only one that proved effective was a capsule containing 1 grain (0.06 gm.) of thyroid extract, 2 grains (0.13 gm.) of suprarenal extract and 2 grains of iron given three times daily. Sajous says that this was "effective," but he does not say restoration of hair occurred, although it is presumed that this is what he meant.

According to Stelwagon⁶ thallium, if given for a long time, will produce universal loss of hair. Buschke and Peiser⁷ fed young rats on bread dipped in a 1 to 5000 solution of thallium acetate, and the rodents lost all their hair in a month. In addition, their growth was stunted and they presented a cretin-like appearance. These writers believe that the endocrine glands showed a selective toxic effect, and the whole organism suffered secondarily through intermediation of the ductless glands. Experiments on tadpoles with thyroid treatment to counteract the thallium have been confirmatory of this belief. In the treatment of epilepsy with borax, Féré⁸ said that it is not rare to see a generalized alopecia produced.

Localized loss of hair may be due to the injury of a nerve or posterior root. In 1886, Max Joseph⁹ demonstrated that section, in an animal, of the posterior branch of the second cervical nerve, between the ganglion and the cord, produced an alopecia in the area supplied by this nerve. This was confirmed by observers in 1887 and 1888.

There undoubtedly is a close relationship between the glands of internal secretion and the sympathetic system, in regard to the growth, character and changes in the hair. Like so many things in endocrinology there are few, indeed, proved facts in regard to the influence of the glands of internal secretion on the sympathetic system and the hair. It is well known that disturbances in the growth and character of the hair occur in certain diseases of the ductless glands, such as myxedema, exophthalmic goiter, Fröhlich's syndrome and

tumors of the suprarenal, but the exact mechanism by which the changes are produced is not known. The hair changes in pregnancy and at puberty are also dependent on the endocrine glands.

Pulay¹⁰ states that at the menopause, either natural or surgical, while the hair falls from the scalp, there is simultaneously an increased growth on the face, and he believes that this is due to cessation of action of the ovaries and decreased function of the pituitary.

C. Todde¹¹ cited the case of an officer, aged thirty-four years, who, following the stress of living at the front and after weeks of incessant bombardment, lost every hair on his body. This man also presented symptoms suggestive of exophthalmic goiter. Todde's case is similar in some respects to our second case.

Meachen and Provis¹² reported an instance of generalized alopecia cured by pregnancy, which relapsed with the re-establishment of the menses. The patient, aged thirty-one years, had been married eight years, and had had four children and two miscarriages. In the sixth month of her second pregnancy the hair began to fall, and when the child was five months of age she "hadn't a hair on her head." During the third pregnancy the hair grew, nearly all of it returning. Then a still birth occurred, and when the periods reappeared the hair fell out again; this time all the hair of the body disappeared. She became pregnant again, and from the first month of the pregnancy the hair began to return, so that she had a splendid crop except in two small patches. When this child was five months of age, the hair once more fell out and the finger-nails also became affected. March 26, 1912, when Meachen and Provis saw her, the scalp was bare, except for a few scanty strands of rather coarse black hair. The eyebrows and eyelashes were absent, although the remaining parts of the body were not absolutely alopecic. The finger and toe nails were lusterless and longitudinally striated, and presented numerous fine pits, especially on the lunulæ. At the time of their presentation of the patient, June 20, 1912, the scalp was totally bald, and the other parts of the body were also denuded. The patient presented no signs of syphilis, but a Wassermann

reaction of the blood had not been taken. In the discussion of this case, Sequeira said that he has seen several cases in which there had been a complete loss of hair after delivery, and he had under his care at that time a woman who had lost her hair on three occasions. The hair returned between the first two pregnancies, but after the third the loss of hair was permanent. He also had under his care at that time a second patient, who had complete alopecia after delivery but when seen twelve months later, the hair was returning.

Eddows¹³ exhibited a patient showing generalized alopecia, leukoderma and scleroderma. The universal alopecia came on after an attack of scarlet fever at the age of eight years. Nothing new occurred until the boy was fourteen, when he awoke one morning with itching hands and shortly after he noticed that his skin was becoming brown. At the time of presentation, the patient was entirely piebald. Large patches of leukoderma were at that time encroaching on all the brown areas. Improvement occurred under the administration of thyroid extract.

REPORT OF CASES

The cases reported here are instructive in the consideration of generalized alopecia:

CASE I.—*History.* A white woman, aged forty-nine years, was admitted to the Episcopal Hospital, March 7, 1923, complained chiefly of general weakness. The family and personal histories were negative, with the exception that eight years before admission to the hospital, she had had a complete hysterectomy. She had never been married or pregnant. The onset of the trouble was one year after the operation (or seven years previously), when the patient began to have severe headaches. At this time the hair of the body commenced to fall out, and in six months all of it had disappeared. Three or four months before admission to the hospital, a few hairs returned on the front of the scalp. Shortly after the appearance of the headaches, she suffered from generalized convulsions every five or six months; in these attacks she was unconscious, bit her tongue, had relaxation of the sphincters, and slept for several hours afterward. The last fit was a year before her admission. During the last year, she had severe frontal headaches, which were sudden in onset, lasted from five to ten

minutes, and were accompanied by nervousness and anxiety. About two months before admission, she had an attack of influenza, from which she made a poor recovery, muscular weakness being so pronounced that she could not walk, and because of this she lost ambition and desired to be left alone. Following the attack of influenza, she was very nervous and so "shaky" that she could not lift a glass of water without spilling it. She had neither pulmonary nor cardiac symptoms and said she had not lost weight.

Examination. The woman was thin and undersized, and lay in bed apparently suffering no pain, although she was nervous and apprehensive. The skin was dry and putty colored. Eight teeth remained, and the tongue was soft and flabby. The lungs, heart and abdomen were normal, except that the right kidney was palpable. All the hair of the body including the eyelashes was absent, with the exception of a small tuft on the anterior portion of the scalp. The deep reflexes, ocular nerves and pupils were normal. No ataxia was present in the finger-to-nose test, although a coarse intention tremor was easily brought out in both upper extremities, especially on the right. Extreme asthenia was present, so that the patient was unable to repeat an act any length of time, in this way resembling a case of myasthenia gravis. The systolic blood-pressure was 95; diastolic, 65. The temperature was subnormal practically throughout the hospitalization. The eye examination by Dr. F. Krauss revealed a primary, bilateral optic atrophy.

The urine was normal, with the exception of an occasional cast and on two occasions a faint trace of albumin; it was reduced in amount, on one occasion measuring 240 cc. in twenty-four hours and on another 775 cc. The routine blood and Wassermann examinations were negative. The blood-sugar was estimated on two occasions; the result of the first examination was 30.42 mg. per 100 cc. and the second, 54.8 mg. The spinal fluid was normal, and the roentgenogram of the skull and sella turcica was negative.

Clinical Course and Outcome. Seventeen days after admission the patient had a severe headache, which was promptly relieved by an ordinary headache powder. Two days later she had a similar headache, and in this became nervous and agitated. On the day before she died she complained of headache, was drowsy and ate practically nothing all day. Spinal puncture was done late on this day, and she died suddenly at 5 o'clock the next morning.

Pathologic and Microscopic Reports. Necropsy was performed five hours after death. The skull was normal, except for a carious condition and enlargement of the sella turcica. The brain and ductless glands were removed for study. The brain was rather

large, especially in its lateral diameter, and the convolutions were flattened. The piaärachnoid was thin and translucent; little vascular sclerosis was present. Bulging of the third ventricle was especially looked for, but could not be made out. On transverse section, it was found that the posterior portions of the lateral ventricles were dilated; the anterior division was filled with a soft, discolored mass not attached to the ventricular walls on the left, but adherent to the under surface of the corpus callosum and the walls of the right lateral ventricle. Both caudate nuclei were compressed. The tumor extended into the right frontal lobe, where it expanded into a mass 3 by 2.5 cm. The right island of Reil was involved and the third ventricle was filled with tumor tissue, but the neoplasm did not extend to the bottom of the third ventricle. Microscopic study of the growth showed it to be a typical glioma. Microscopic study of the tuber cinereum revealed a distorted but comparatively normal structure.

The pituitary, examined by Dr. E. B. Krumbhaar, was large; acidophilic hyperplasia was noted in the center, with a cyst containing degenerated cells and coagulated material, lined with tall columnar epithelium, with invasion of round-cell nuclei in the immediate vicinity. Small hemorrhages could be made out. The suprarenal was in the resting stage and showed slight interstitial irritation. The pancreas was the seat of moderate fatty infiltration. The thyroid, parathyroids and pineal were normal.

CASE II.—A white man, aged thirty-one years, stated that he first observed a gradual loss of hair over the entire body after undergoing some harrowing experiences in France during the war, and within a few months the entire body was denuded of hair with the exception of the eyelashes and one or two small, fine hairs on the upper lip; an increase in weight was also noted.

When we saw him, five years later, the patient presented the picture of a complete alopecia, with the exception of the eyelashes (Figs. 1 and 2). In addition the skin was soft and velvety; the breasts were slightly enlarged and he was 20 pounds (9 kg.) above his usual weight. The gonads were small. In all other respects, the general physical examination was negative. A roentgenogram of the sella turcica was negative, and the blood chemical findings were within normal limits, the blood-sugar being 80.

CASE III.—*History.* A waitress, aged thirty-three years, unmarried, was admitted to the Episcopal Hospital, May 21, 1925, born in Norway, had been in the United States sixteen years. The

past history was unimportant. The menses were normal and she had never been pregnant.

Her chief complaint was loss of hair over the entire body. In August, 1923, she noticed some bald spots on the back of her head. By Christmas of that year, all the hair had disappeared from the head, eyebrows, eyelashes, and the hair from the rest of the body disappeared soon afterward. She had lost 40 pounds (18 kg.) since the onset of the trouble. She had had various lines of treatment, but nothing seemed to cause a return of hair.

Examination. The patient was tall, angular and emaciated. Physical and neurologic examinations were entirely negative, except that all the hair of the body was absent (Figs. 3 and 4). The roentgenogram of the sella turcica revealed no evidence of abnormality. The eye-grounds and fields were normal. The temperature showed little if any deviation from the normal throughout the stay in the hospital. The urine and spinal fluid were normal. The blood chemical findings and blood Wassermann reaction were negative. The blood count was not unusual, with the exception of an eosinophilia of 6 per cent, for which no cause could be found.

A piece of scalp was removed and submitted to Dr. Fred D. Weidman, professor of dermatologic research at the University of Pennsylvania, who found that the plane of excision from the scalp had passed through the deepest parts of the corium.

Epiderm: The uppermost parts of the stratum corneum were loose, but the layer as a whole was thin. The granular layer was likewise thin, and its cells appeared normal. The prickle layer averaged four or five layers of large, normal-appearing cells with large spherical nuclei. The intercellular spaces were normal, prickles being recognizable only on careful search. The basal layer consisted of only one or two layers of cells; these were not quite normal in appearance, because they conformed so closely to those of the prickle layer. Thus, they averaged larger in size; the nuclei were large and not particularly elongated, and the chromatin was not condensed. The interpapillary pegs were, if anything, less highly developed than normal. On the whole, the epiderm seemed to be, clinically speaking, thin, soft and supplied with a full amount of watery material, the latter, of course, bound up with the protein molecule (hydrophil colloid).

Corium: The pars papillaris appeared normal. The collagen bundles of the deeper parts took on the appearance of being separated into sectors by reason of the looseness of perivascular, perifollicular and perisudoriferous fibrous tissue. In the latter positions, the spindle cells were swollen and notably hyperplastic;

only small numbers of lymphocytes were intermixed. The blood-vessels (capillaries were the only ones included in this section) were numerous, and appeared in these sections in clusters. Dr. Weidman did not believe that this was the effect of tortuosity but rather of multiplication. Their lining cells were plump and hyperplastic. As indicated above, they were surrounded by a loose reticulum, on which goodly numbers of swollen spindle cells were disposed.

Hair Follicles: These generally occurred in groups of three or four. They were numerous. The most conspicuous feature consisted in the absence of the hair shaft; in its stead a loose, pink, fibrillar material appeared, which was identical in appearance with the corneous substance on the skin surface. The sheath consisted on an average, of eight or ten layers of cells, and was a replica of the surface epiderm. Thus, those parts of the follicle which lay closer to the surface exhibited a most sharply and fully developed stratum granulosum; if anything, a basal layer was better indicated here than on the surface. However, no prickle cells could be made out. In deeper portions of the follicle, there was a delicate, thread-like, hyaloid (but doubtless keratinous, chemically) zone lining the lumen of the hair follicle. Its wavy contour reminded one of the elastic membrane in arteries and perhaps of the cuticle-like lining of sweat ducts. It is regretted that none of the bulbs of follicles were included in these sections.

Sebaceous Glands: These were most inconspicuous for a position like the scalp, although they might have been more fully represented if deeper portions had been available for study. As it was, only small groups of cells adjacent to the hair follicle could be found. Those that were exhibited appeared normal.

Sweat Glands: Again, since the examination was restricted to the upper portions of the scalp, the deeper parts, such as the secreting coils, were not represented. Ducts, however, were numerous. The lumina were narrow and their lining cells plump and of normal appearance.

Comment and Summary: No exceptions could be taken against the epiderm; if anything, it appeared to be biologically more active than normal. The important changes were those of the hair shaft, together with the assumption of epiderm-like qualities on the part of the sheath. The second significant feature was the hyperplasia of the capillaries and of the spindle cells around them; but there was nothing to indicate whether this was a primary or secondary factor in the case. The same was true of the scarcity of sebaceous glands, but in this case the explanation was at hand in the form of the secondary atrophy of disuse.

Supplementary Note: In subsequently cut sections, the lowermost portions of some hair follicles were presented for study. Now the whole follicle was less than half the size of those elsewhere, and easily overlooked. The peripheral parts of the sheath merged with the surrounding loose fibrous tissue by reason of an edema, which affected both of these members. The sheath consisted of only two or three layers of cells, the basal ones not being recognizable as such. The center of the follicle consisted of some pink, probably keratinous substance, enclosing numerous epithelial nuclei. The latter were degenerate—some were swollen and pale, while others were shriveled, solid and almost black. Small numbers of lymphocytes and one or two polymorphonuclears appeared in the neighborhood of these follicles. The appearance was not that of an inflammation and fibrosis which was inducing an atrophy of the follicle, but more that of a primary atrophy. The surrounding loose fibrous tissue and minor cellular infiltrate might be interpreted as reactions to disintegration products of the hair shaft that were once present.

The histologic diagnosis was atrophy of the hair follicle and sebaceous glands and "epidermization" of hair follicle.

SUMMARY AND CONCLUSIONS. These three cases are examples of generalized alopecia. In the first, the condition came on one year after a complete hysterectomy, with the coincident occurrence of symptoms that were due to brain tumor. The fact that removal of the ovaries usually produces hypertrichosis and also that the alopecia did not occur until one year later would militate against the possibility that the condition was ovarian in origin. The situation of the brain tumor directly over the pituitary and in the third ventricle produced erosion of the sella turcica which, with the low blood-sugar and hypotension, was suggestive of a disturbance of function in the region of the infundibulum or pituitary, or both.

In the second case, the symptoms came on after severe emotional stress and here, too, there are symptoms referable to the pituitary region, although not necessarily a tumor. It may be said by some that emotional stress is incapable of producing such a marked disturbance of a ductless gland, yet we have seen exophthalmic goiter develop in a woman shortly after she had nearly drowned. Furthermore, the so-called neurocirculatory asthenia or effort syndrome which in certain cases may be of endocrine origin occasionally comes on after

severe fright, and in this group of cases disability persists for many years. Other cases of universal alopecia following emotional stress have been recorded; thus, Sabouraud¹⁴ described a case of profuse alopecia in a girl, aged thirteen years, who lost her hair thirteen days after being raped, although the hair returned later. Boisser, Bidon and Morris¹⁵ have all cited cases of generalized alopecia coming on after severe emotional stress.

The cause of the alopecia in the third case is unknown to us. In this case, as in the second one, we tried various glandular extracts over a period of one year but without results.

REFERENCES.

1. Chambers, G.: Alopecia Universalis, Jour. Am. Med. Assn., 1901, xxxvi, 57.
2. Arnett, U. G.: Am. Jour. Syph., 1922, vi, 131.
3. Thompson, L. O.: Syphilis, 2d ed., Philadelphia, Lea & Febiger, 1920, p. 112.
4. Alfred, A. R.: U. S. Navy Med. Bull., 1919, xiii, 539.
5. Sajous: Internal Secretions and Principles of Medicine, Philadelphia, F. A. Davis Company, 1920, ii, 1827.
6. Stelwagon, H. W.: Diseases of the Skin, Philadelphia, W. B. Saunders Company, 9th ed., p. 1047.
7. Buschke, A., and Peiser, B.: Med. Klin., 1922, xviii, 731.
8. Féré: Compt. rend. Soc. de biol., 1893, v, 987.
9. Joseph, M.: Presse méd., 1922, p. 855.
10. Pulay, E.: Med. Klin., 1922, xviii, 1369.
11. Todde, C.: Riforma méd., 1920, xxxvi, 382.
12. Meachen and Provis: Brit. Jour. Dermat., 1912, xxiv, 272.
13. Eddows: Brit. Jour. Dermat., 1899, xi, 325.
14. Sabouraud, R.: Presse méd., 1923, xxxi, 14.
15. Boisser, Bidon and Morris, quoted by Stelwagon: Diseases of the Skin, p. 1048.

MULTIPLE NEURITIS FOLLOWING CARBON MONOXID POISONING

A CLINICOPATHOLOGIC STUDY*

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IN recent years quite a number of contributions to the literature have appeared describing the changes in the central nervous system which result from carbon monoxid poison. The most constant of all these pathologic findings is bilateral softening of the globus pallidus. Cortical changes, while not nearly so common, have been described.¹ Multiple neuritis as a sequel to gas poisoning has been reported clinically, but satisfactory pathologic examination of the peripheral nerves is practically unknown. Starr,² Jelliffe and White,³ Krantz,⁴ Brissaud,⁵ Lancereaux⁶ and Glynn⁷ have cited clinical cases of multiple neuritis secondary to carbon monoxid poisoning. Stewart¹ reported that the seventh and tenth nerves in a case of gas poisoning were intensely degenerated, this being especially true of the tenth. He did not examine nerves from the extremities.

Claude and Lhermitte⁸ tried to produce multiple neuritis in dogs by the use of carbon monoxid. They found that they could produce small hemorrhagic areas in the cortex and in the cord, but the peripheral nerves remained intact. By the simultaneous use of another poison (diphtheria toxin), the peripheral nerves became involved. Claude and Lhermitte concluded that peripheral neuritis was not produced by carbon

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monoxid *per se*, but occurred only when another poison was used in conjunction with it.

Maczkowski⁹ explained the cause of the neuritis from carbon monoxid poisoning as the result of trauma or blood extravasation, due to changes in the vessel walls or to alteration of the chemistry of the blood.

Brissaud stated that this form of multiple neuritis is atypical because the deep reflexes are usually increased. While Brissaud did not give a reason for this, the increased reflexes are due, in all probability, to involvement of the globus pallidus or of the cortex or of both.

Remak and Flatau¹⁰ say that a typical multiple neuritis following carbon monoxid poisoning has not been described, although localized neuritis is not uncommon. An extensive bibliography is given in their article.

We report 3 cases, 2 with necropsy, of patients who had evidences of polyneuritis occurring as a sequel to gas poisoning.

CASE I.—L. C., a woman, aged fifty-four years, brought to the Episcopal Hospital by the police, April 3, 1923, was unconscious, having been found stuporous in her room with the gas turned on. The length of time she was exposed to illuminating gas was unknown. She was well nourished and developed. She was perspiring freely and was completely unconscious. Her cheeks were markedly flushed and respiration was rapid and labored. The pupils were equal, round and regular, and widely dilated; they reacted poorly to light. Examination of the lungs showed numerous coarse râles throughout. The heart was enlarged; the rate was rapid, but the rhythm was regular, with accentuation of the second aortic sound. The deep reflexes were all present and active. Plantar stimulation produced an extension of the great toe on both sides, but the reflex was not atypical. The Babinski reflex, ankle clonus and Kernig's sign were not present.

A few days after admission, she improved and answered questions, but she was disoriented as to time and place. A week after admission, the right hand and forearm became cold and pale, and a pulsation could not be felt in either the radial or the brachial artery. Tenderness was present on squeezing the nerve trunks of the extremities, although the deep reflexes remained prompt. The condition of the right upper extremity progressed until the hand became blue and reddish purple. The patient became unconscious, April 13, and died the following day.

Transverse and longitudinal sections were studied from the ulnar, median, brachial plexus, plantar, popliteal and sciatic nerves on each side. These sections were stained by the Marchi method alone (Figs. 1 and 2), and also counterstained by the Alzheimer-Mann, Mallory-Jacob and Bielschowsky stains. Thin sections in paraffin were also stained with the Bielschowsky and Mallory-Jacob methods. The Marchi sections showed a marked increase of Elzholz bodies, which tended to localize, leaving portions of the nerve fiber free (Fig. 3). The medullary sheath was swollen and tumefied, showing in places large spaces (lücken) through which the intact axis cylinder could be seen passing (Fig. 4). Only rarely was there axis cylinder change, and when present it consisted of a slight fusiform swelling. No change was detected in the sheath nuclei. Despite the fact that the right axillary artery was thrombosed, the nerves from that extremity were not more involved than those from the opposite extremity. The nerves from the upper extremities were more involved than those from the lower limbs.

In the spinal cord, no tract changes were present. The anterior horn cells were lessened in number and stained poorly (Fig. 5), and some of them showed axonal chromatolysis and lipoid accumulations (Fig. 6).

CASE II.—A man, aged sixty-eight years, admitted to the Philadelphia General Hospital, January 28, 1924, had been found unconscious in a small room heated by charcoal. He could not be aroused, and was taken to the hospital unconscious. He had an ischiorectal abscess, for which he had been treated. His skin was cold and dry, with a cherry-red tint. Respirations were labored and stertorous, and the breath had a sweetish, musty odor. On admission, the lungs and heart presented no abnormality. Generalized muscular twitchings were noted; some involved the entire muscle and others apparently only the muscle fiber. The bladder was distended almost to the umbilicus. Examination of the anal region revealed evidence of an ischiorectal abscess, which was draining freely. Shortly after admission, the man had an epileptiform attack, which was followed by coma. The pupils were small and reacted poorly to light. The nasal side of each disk was indistinct and the veins were overfilled. All the deep reflexes were present and active, without clonus or Babinski's or Kernig's sign. All the extremities were moved, but apparently with difficulty. A marked degree of muscular rigidity was present; this was increased by passive movement.

The patient lived eleven days. During the last few days of

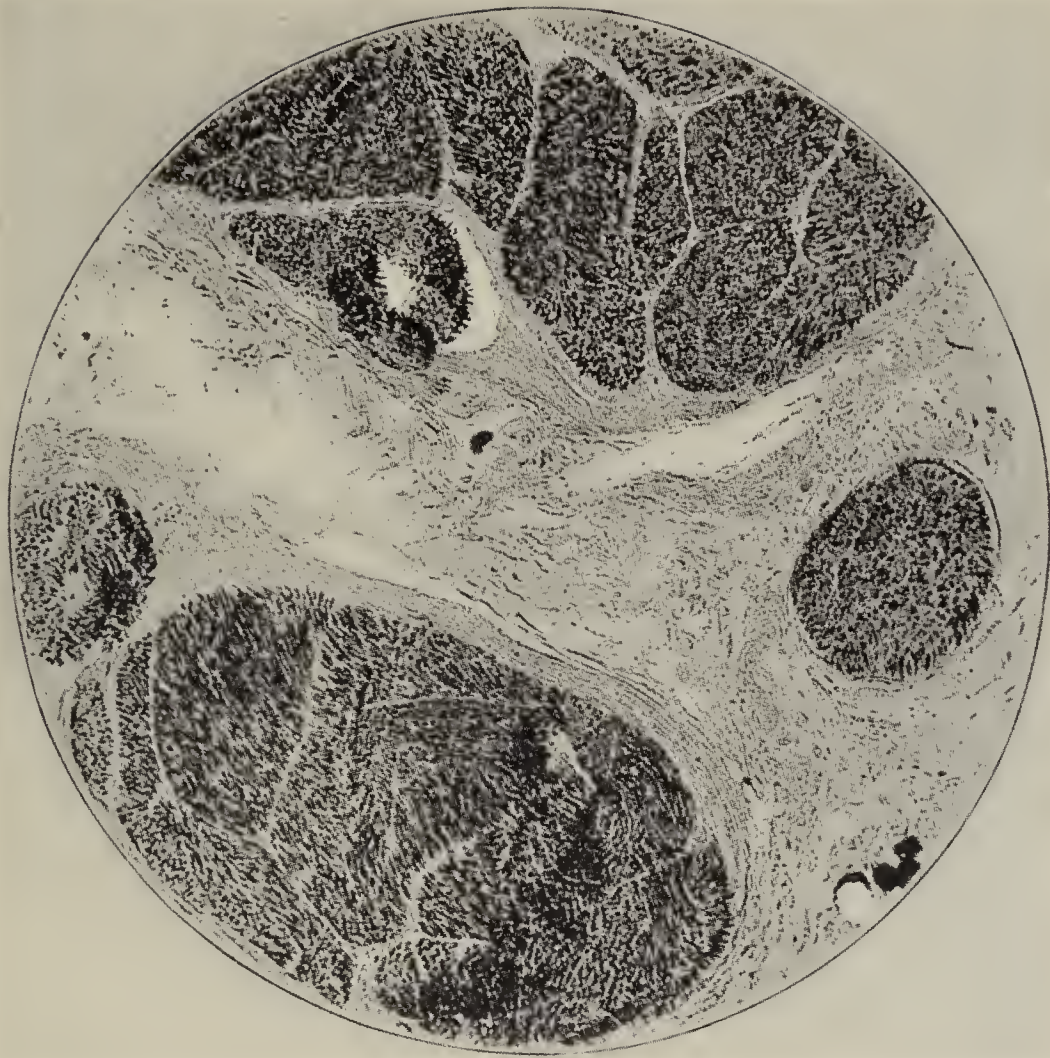


FIG. 1.—Transverse section of right median nerve, showing widespread involvement; Marchi stain.

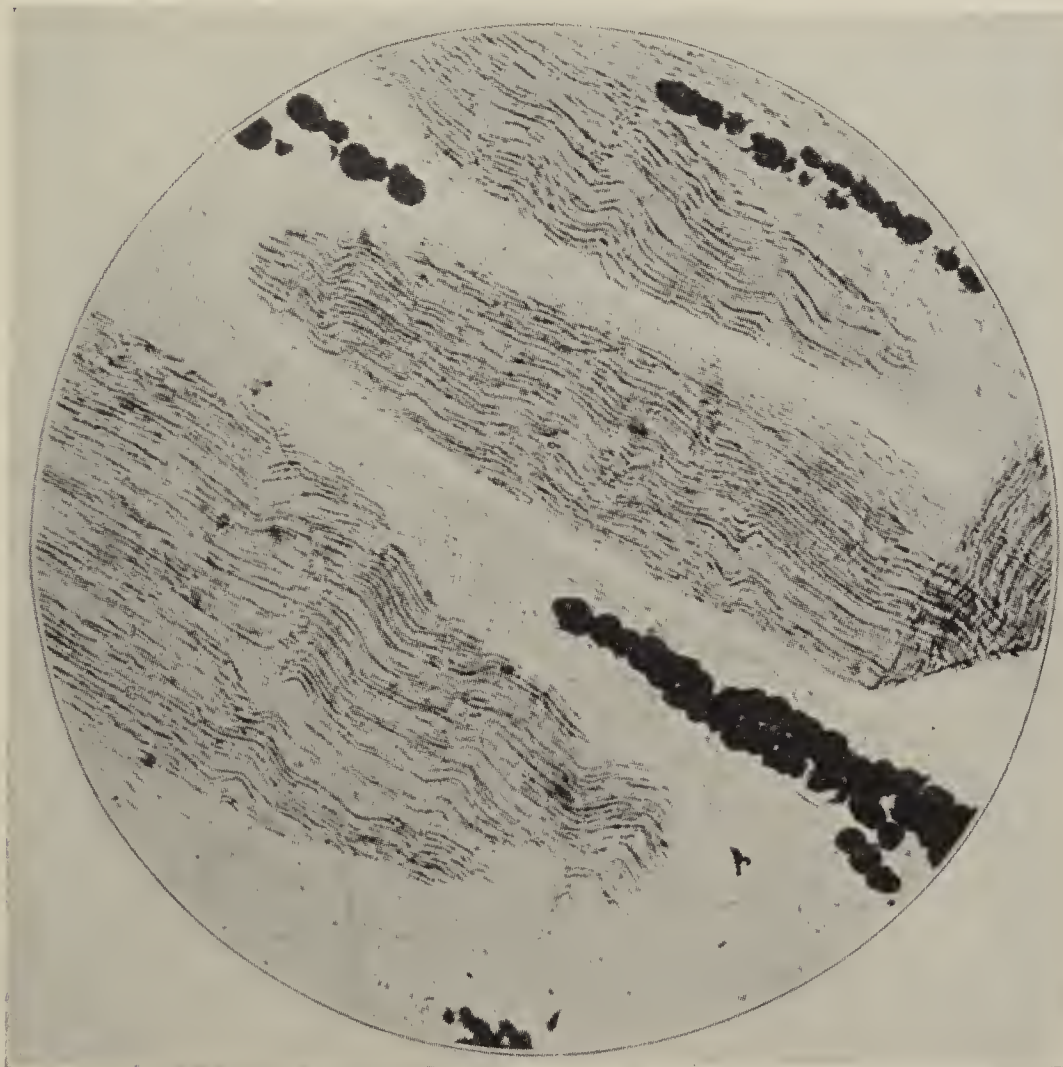


FIG. 2.—Longitudinal section of right external popliteal nerve showing marked degeneration; Marchi stain.

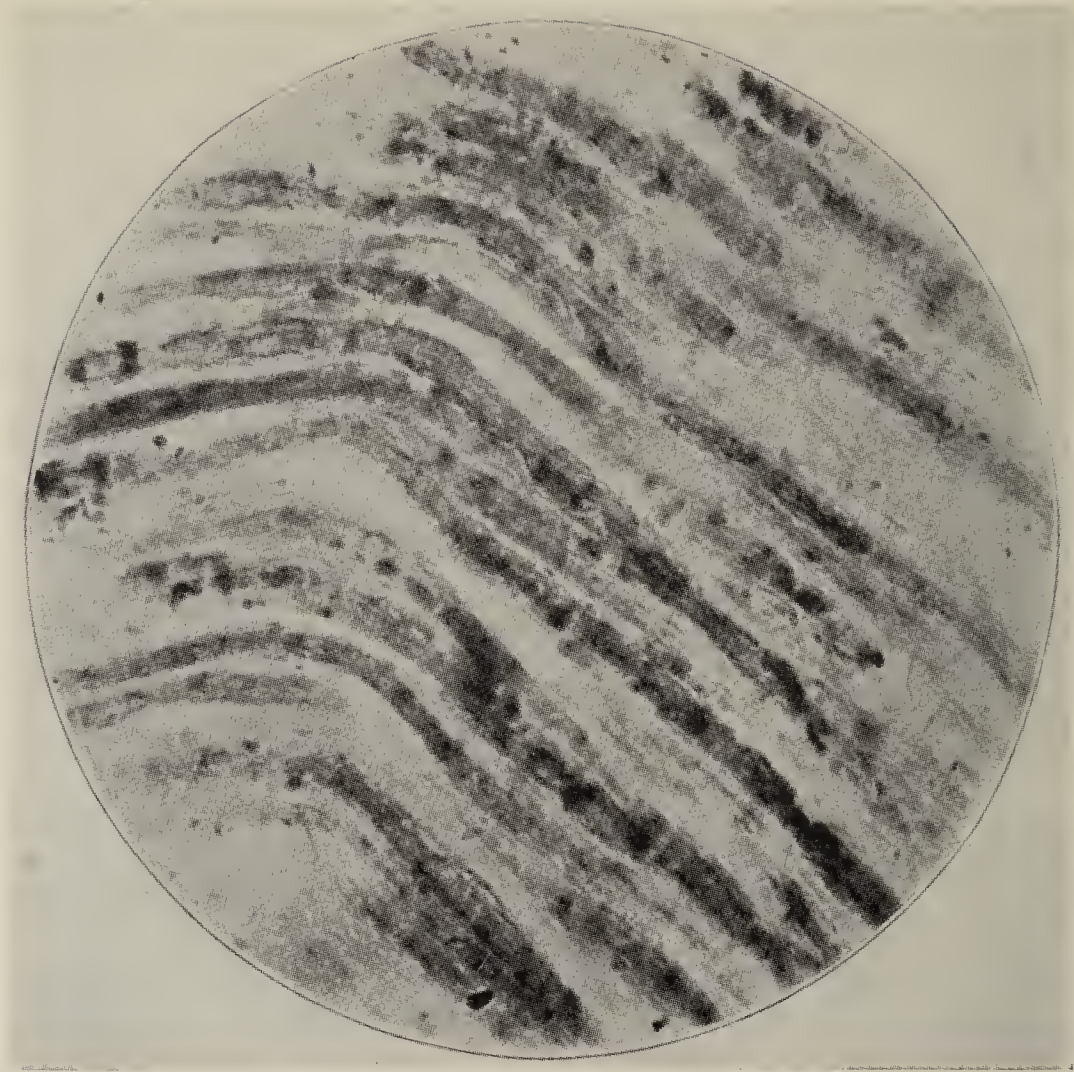


FIG. 3.—Same section as Fig. 2 with higher magnification; Marchi stain.

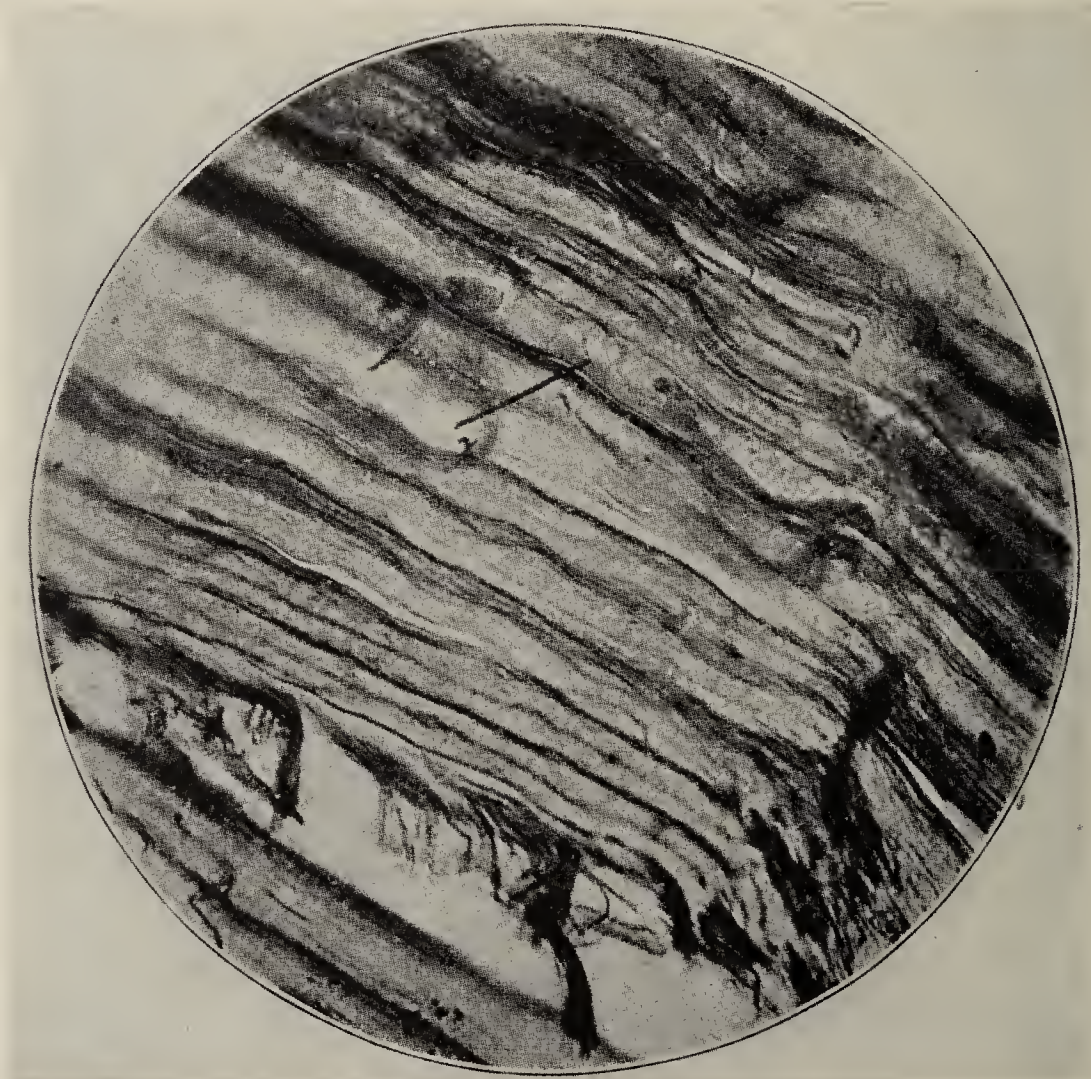


FIG. 4.—Longitudinal section of left ulnar nerve showing normal axis cylinders and swollen myelin sheaths; phagocytic cells within one fiber at x; Mallory-Jacob stain.

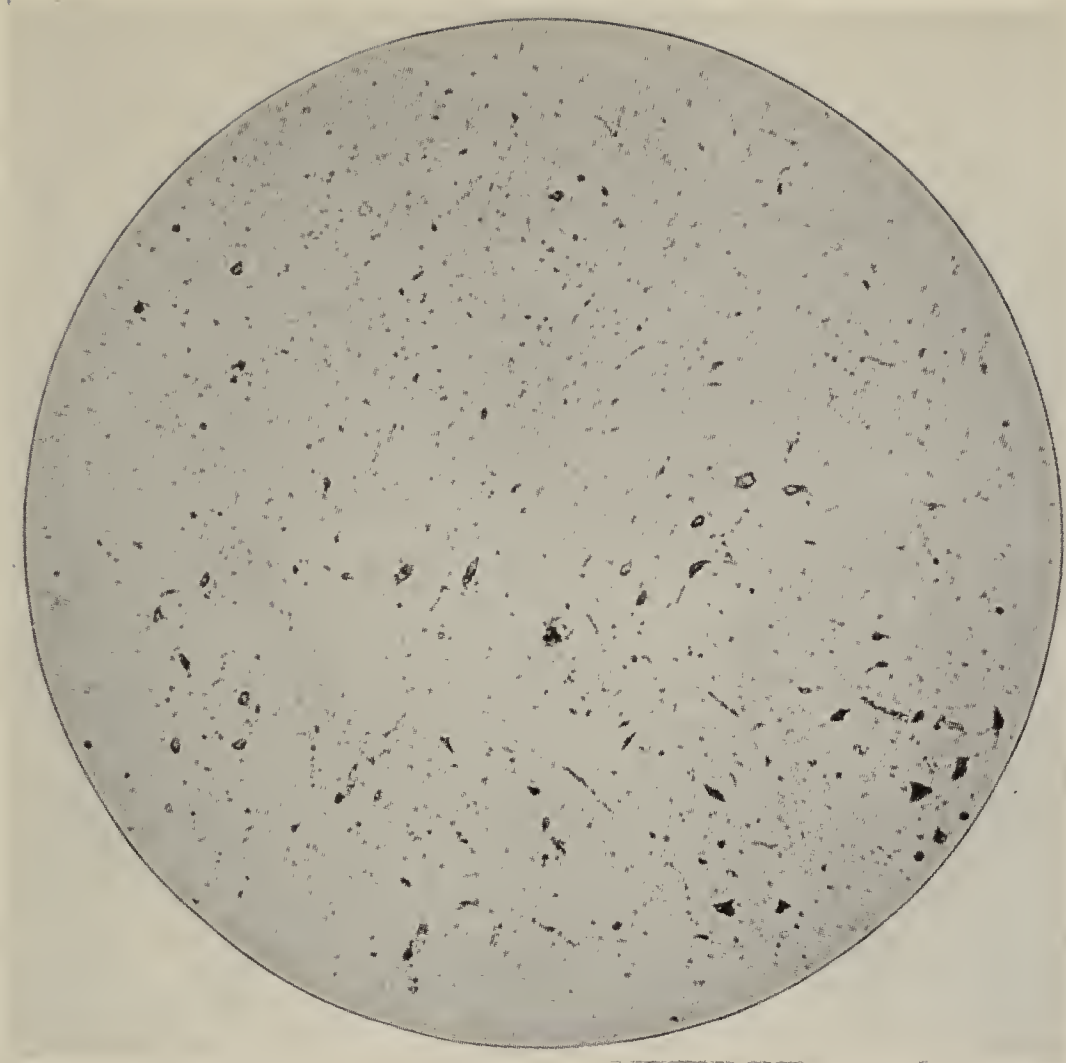


FIG. 5.—Anterior horn cells of lumbar cord showing diminution in number and poor staining qualities of cells; toluidin blue stain.



FIG. 6.—Two cells from anterior horns showing axonal chromatolysis at *a* and lipoid accumulation at *b*.



his illness the temperature ranged from 102° to 104° , with greatly increased respiratory and cardiac rates; the cause of this condition was bronchopneumonia, which was the immediate cause of death. The right side became very rigid, whereas the left arm and leg presented spasticity and myoclonic movements identical to those seen in epidemic encephalitis.

Spinal puncture was performed; the fluid was negative on examination. Four days after his admission to the hospital a swelling of the left knee-joint developed; two days later, this was tapped and a purulent fluid obtained. Examination of the fluid showed no organisms and a culture also was negative. A coagulum formed in the fluid from which a smear showed a considerable number of degenerated cells, of which 70 per cent were polymorphonuclears and the remainder lymphocytes.

On the fifth day of his illness a note was made that the patient developed persistent hiccuping and that the face was masked. Muscle rigidity persisted, and movements of the extremities produced pain.

Histologic examination of the peripheral nerves, spinal ganglions and muscles revealed that the pathologic changes in this case corresponded very closely to those seen in the preceding one, with the exception that there was not a uniform involvement of the peripheral nerves. In this case some were intensely degenerated, some moderately and others not at all. Marchi's stain showed an increase of Elzholz bodies. When counterstained with Mallory-Jacob and Alzheimer-Mann stains, the nerve fibers were seen to be swollen, but the axons suffered no great damage. Occasionally a nerve fiber with a vacuolated sheath was seen. Sections of the muscle showed no alteration.

Examination of the spinal ganglions showed but little change in the fibers entering and emerging, but the nerve cells themselves showed an increase of fat content.

CASE III.—L. S., a blacksmith, aged fifty-one years, admitted to the Philadelphia General Hospital, March 18, 1919, had been overcome by illuminating gas, January 10, when a man in the room next to his had committed suicide by gas and enough of the gas had entered the subject's room to render him unconscious. He had been taken to a nearby hospital and regained consciousness in a few hours; he had been discharged the same day. A few days later, he began to have difficulty in swallowing, and food or liquids that did enter the stomach were promptly vomited. The vomiting ceased a week later, but the dysphagia persisted.

A week after he had been poisoned by gas, pain appeared in the feet and legs. The pain at first was of a burning character and was increased by walking and by pressure.

On examination, the man lay in bed, groaning and moaning continuously. The lips, nose and ears were moderately cyanosed. The pupils were contracted, equal and regular, and reacted well to light and in convergence. The cranial nerves were normal except for a spasm of the motor fifths. The arms were moved freely in all directions, and showed a coarse tremor which was not intensified by action. The biceps and triceps reflexes were present and exaggerated. There was tenderness on pressure over the nerve trunks and over the brachial plexuses. Movements of the lower extremities were performed normally, with the exception of extension of the feet and toes. These movements were practically absent. The knee-jerks were present and exaggerated; the Achilles jerks were absent. There was great tenderness on pressure over the nerve trunks of the lower extremities and on lateral squeezing of the feet. Plantar stimulation produced no response on either side. The arteries and heart were in good condition. The systolic blood-pressure was 120; diastolic, 85. The chest was round and the lungs emphysematous. At the right apex there were signs of a limited, quiescent tuberculous condition.

A roentgen-ray examination of the esophagus and stomach, to determine, if possible, the cause for the dysphagia and vomiting, revealed the presence of cardiospasm.

The urine was normal with the exception of an occasional granular cast. The spinal fluid and blood presented no abnormalities.

During the first week of the man's stay in the hospital, he improved in a general way, although the polyneuritic state remained unchanged. A stomach tube was passed without trouble, showing that there was no obstruction. He could now swallow solid food, and lived entirely on milk and soup.

During the last three weeks of his life he became progressively worse, and, owing to lack of sufficient food, he lost weight. The temperature, which was normal for ten days after admission, began to show a distinct upward trend, and toward the close was continuously above 101° ; the cause of the elevation was a bronchopneumonia, which may have been tuberculous, although tubercle bacilli were not found in the sputum on repeated examinations.

The upper and lower extremities became spastic, and at times were difficult to move. When the patient reached for objects a tremor developed; the tremor changed in character toward the close, and was of an intention type.

The face was expressionless, and at times the jaws could not be forcibly opened. The lower extremities were spastic at the knees, and showed a flaccid paralysis of the feet, with exaggerated patellar and lost Achilles reflexes. The plantar reflexes were not present at any time of the patient's illness. The abdominal reflexes also were absent. The tenderness over the nerve trunks persisted, and toward the termination of the illness, when the man was difficult to arouse, pressure over the large nerves produced discomfort and caused outbreaks of crying and moaning.

Two subsequent lumbar punctures were done. The fluid was acellular, contained no organisms and showed a negative Wassermann reaction. Death occurred, April 16. No necropsy was obtained.

COMMENT. These patients had clinical evidence of multiple neuritis, although the picture was unusual in that the deep reflexes were increased. The exaggeration of the deep reflexes, as we have stated above, is due in all probability to the involvement of the globus pallidus or of the cortex or of both. The person who has a combination of conditions—disease of the basal ganglions or cortex and of the peripheral nerves—will show either no alteration or an exaggeration of the deep reflexes, unless the peripheral nerves are greatly affected. We may mention, as analogous conditions, the increase of the deep reflexes in alcoholic multiple neuritis, in which there is in addition cerebral involvement; also the increase of the deep reflexes in disease of the spinal cord, in which the pyramidal tracts are involved in conjunction with disease of the anterior horns or of the posterior columns. In Case III, in which unfortunately no necropsy was obtained, a paradoxical condition was found in that there was spasticity and increase of the deep reflexes at the knees, while at the ankles flaccidity and lost reflexes were found. In Case I the changes in the peripheral nerves were distinct; in Case II the findings were distinct, but not so pronounced.

We believe that multiple neuritis occurring as a sequel to carbon monoxid poison is probably much more frequent than is commonly believed, yet direct pathologic evidence of such an involvement is practically unknown in the literature. The peripheral nerves are not often removed at necropsy, and this

is probably why changes in these parts are not more frequently discussed.

We are indebted to Dr. A. A. Stevens for the privilege of reporting Cases II and III.

REFERENCES

1. Stewart, R. M.: *Jour. Neurol. and Psychopath.*, 1921, i and ii, 125.
2. Starr: *Nervous Diseases Organic and Functional*, Philadelphia, Lea & Febiger, 1913, p. 222.
3. Jelliffe, S. E., and White, W. A.: *Diseases of the Nervous System*, Lea & Febiger, Philadelphia, 1923.
4. Krantz, Joseph: *Aus Aachen, Ludwig Maximilians Universität zu München*, 1913.
5. Brissaud: *Paralyses toxiques*, Paris, Asselin and Houzeau, 1886, p. 138.
6. Lancereaux: *Anatomie pathologique*, Paris, Delahaye, 1889, iii, 358.
7. Glynn: *Brit. Med. Jour.*, 1895, i, 759.
8. Claude and Lhermitte: *Compt. rend. Soc. de biol.*, February 3, 1912, p. 164.
9. Maczkowski: *Neurol. Centralbl.*, 1900, xix, 520.
10. Remak and Flatau, in Nothnagel: *Spezielle Pathologie und Therapie*, 1900, ii, 627.

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